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LEMPERT DECOMPRESSION OPERATION FOR HYDROPS OF THE ENDOLYMPHATIC LABYRINTH IN MÉNIÈRE'S DISEASE

JULIUS LEMPERT, M.D.
NEW YORK

MÉNIÈRE'S DISEASE, the cause of which is still to be determined, is diagnosable only through the process of exclusion. It consists of repeated sudden, severe attacks of vertigo with spontaneous nystagmus occurring at intervals of unpredictable frequency and duration. The attacks are accompanied with severe tinnitus, in addition to a loss of perceptive hearing which usually progresses with each attack. If allowed to continue, the paroxysmal attacks usually result in complete inner ear deafness. As a rule, the vestibular function when tested between attacks shows diminished response as compared with that of the nonaffected ear. These attacks are usually accompanied with nausea and vomiting.

PATHOLOGIC ASPECTS

In 1938 Hallpike and Cairns[1] first observed a hydrops of the vestibular and cochlear portions of the membranous labyrinth on histologic examination of the temporal bone of the affected ear in 2 cases of Ménière's disease which came to autopsy. In 1940 Hallpike and Wright[2] were able to make the same observation in another case. These observations have been confirmed by Lindsay[3] in 1942 and by Altman and Fowler Jr.[4] in 1943.

On the basis of these histologic observations of hydrops occurring within the labyrinths of patients known to have suffered from Ménière's disease, it became logical to reason that the signs and symptoms of Ménière's disease are most likely caused by this hydrops of the endolymphatic labyrinth. The etiologic factors precipitating such hydrops are still to be determined

From the Lempert Research Foundation, Inc.

1. Hallpike, C. S., and Cairns, H.: J. Laryng. & Otol. **53**:625, 1938.
2. Hallpike, C. S., and Wright, A. J.: J. Laryng. & Otol. **55**:59, 1940.
3. Lindsay, J. R.: Labyrinth Dropsy and Ménière's Disease, Arch. Otolaryng. **35**:853 (June) 1942.
4. Altman, F., and Fowler, E. P., Jr.: Ann. Otol., Rhin. & Laryng. **52**:52, 1943.

SURGICAL TREATMENT OF MÉNIÈRE'S DISEASE

Both the clinical picture and the histologic aspects of Ménière's disease permit the conclusion that it is a disease limited entirely to the internal ear and that both the vestibular and the cochlear portion thereof are involved.

It is therefore logical to assume that by effecting the complete degeneration of both the vestibular and the cochlear part of the endolymphatic labyrinth in a case of monaural Ménière's disease, and thus preventing the recurrence of endolymphatic hydrops, it should be possible permanently to relieve the patient of the vertigo, which is vestibular in origin and the tinnitus, which is most likely of cochlear origin. A treatment of Ménière's disease cannot be considered completely adequate unless it relieves both of these distressing symptoms.

DOES PARTIAL DESTRUCTION OF THE VESTIBULAR PORTION OF THE MEMBRANOUS LABYRINTH RESULT IN DEGENERATION OF THE ENTIRE ENDOLYMPHATIC SYSTEM?

It is a well recognized fact today that partial section or electrocoagulation of the vestibular portion of the endolymphatic labyrinth through a fistula created in the external semicircular canal, though as a rule resulting in cessation of the paroxysmal attacks of vertigo, seldom relieves the tinnitus. Believing as I do that the complete degeneration of both the vestibular and the cochlear portion of the endolymphatic labyrinth should prevent the recurrence of endolymphatic hydrops and result in permanent relief of both the vertigo and the tinnitus in Ménière's disease. I could not help but question whether degeneration of the entire vestibular and cochlear endolymphatic labyrinth is ever accomplished by partial destruction of the vestibular portion of the labyrinth.

In my pursuit of a research study of this problem, my attention was attracted by the clinical observation that when the external semicircular canal of the endolymphatic labyrinth was accidentally destroyed or removed during the performance of the fenestration operation for clinical otosclerosis the accident did not always result in a still further recession of the preoperatively impaired hearing. Such observation strengthened my belief that partial vestibular labyrinthectomy does not necessarily always result in degeneration of the entire endolymphatic system.

However, not until I carefully studied histologically the internal ears of our experimentally fenestrated rhesus monkeys[5] was I convinced that destruction of the vestibular portion of the labyrinth when performed under sterile precautions did not result in degeneration of the rest of the endolymphatic labyrinth and of the organ of Corti.

5. Lempert, J.; Meltzer, P. E.; Schall, L. A., and Wolff, D.: Osteogenesis Following Fenestration of the Vestibular Labyrinth of the Rhesus Monkey: A Controlled Experimental Study, Arch. Otolaryng. **46**:512 (Oct.) 1947.

Figures 1 to 5 show photomicrographs of the endolymphatic systems of the experimentally fenestrated labyrinths of these rhesus monkeys, proving that destruction of the vestibular portion of the mem-

Fig. 1.—Normal cochlea in the presence of destruction of the crista of the semicircular canal and obliteration of the lumen of the lower part of the canal (as seen in the extreme right of the lower picture) following fenestration. Experiment II; monkey 24, right ear; eight months and two weeks after operation.

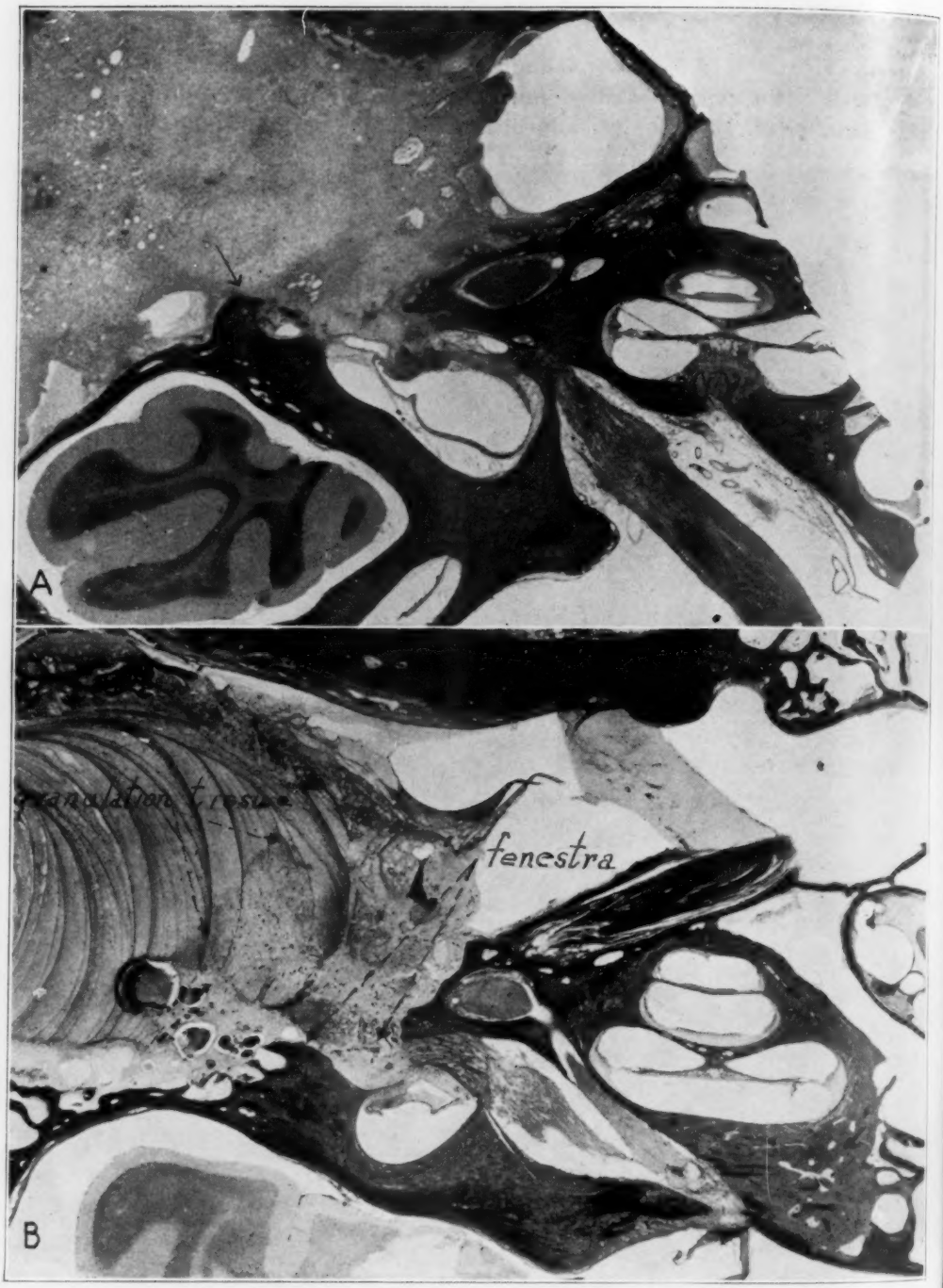

Fig. 2.—*A*, destruction of the crista of the horizontal canal by a large mass of mature bone lost within the perilymphatic space during fenestration. Note the normal cochlea with Reissner's membrane in situ and taut. The organ of Corti is in excellent state. Experment I; monkey 15, left ear.

B, complete destruction of the crista of the horizontal canal with loosening of the perilymphatic tissue from the endosteal wall of the ampulla. A sagging of Reissner's membrane is the only pathologic change noted in the cochlea. Experiment I; monkey 11, left ear; six days after operation.

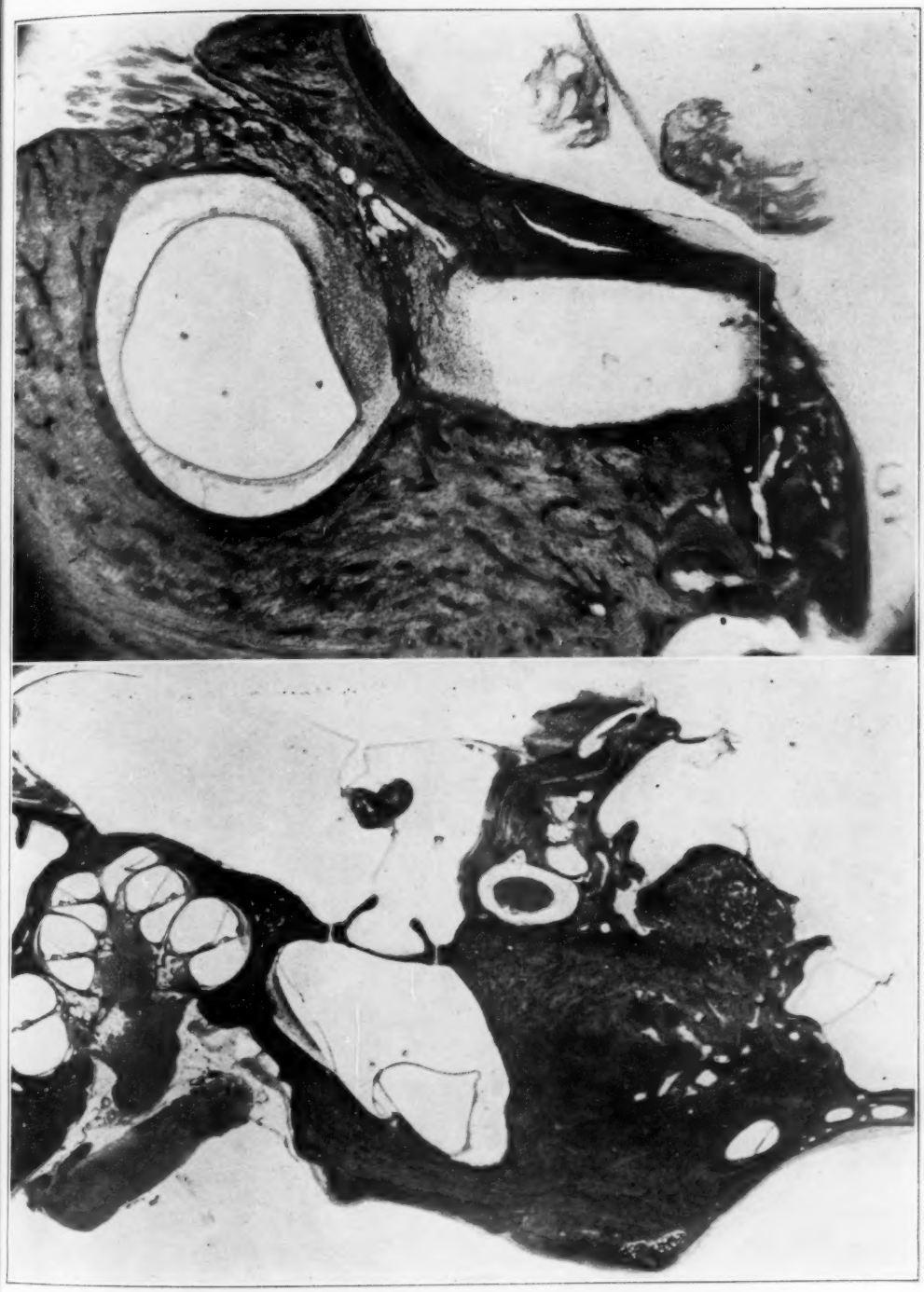

Fig. 3.—Fenestra in external semicircular canal closed as a result of periosteal bone regeneration. The membranous labyrinth and the endosteum were destroyed during fenestration. The cochlea remained in excellent condition. Monkey 59, right ear.

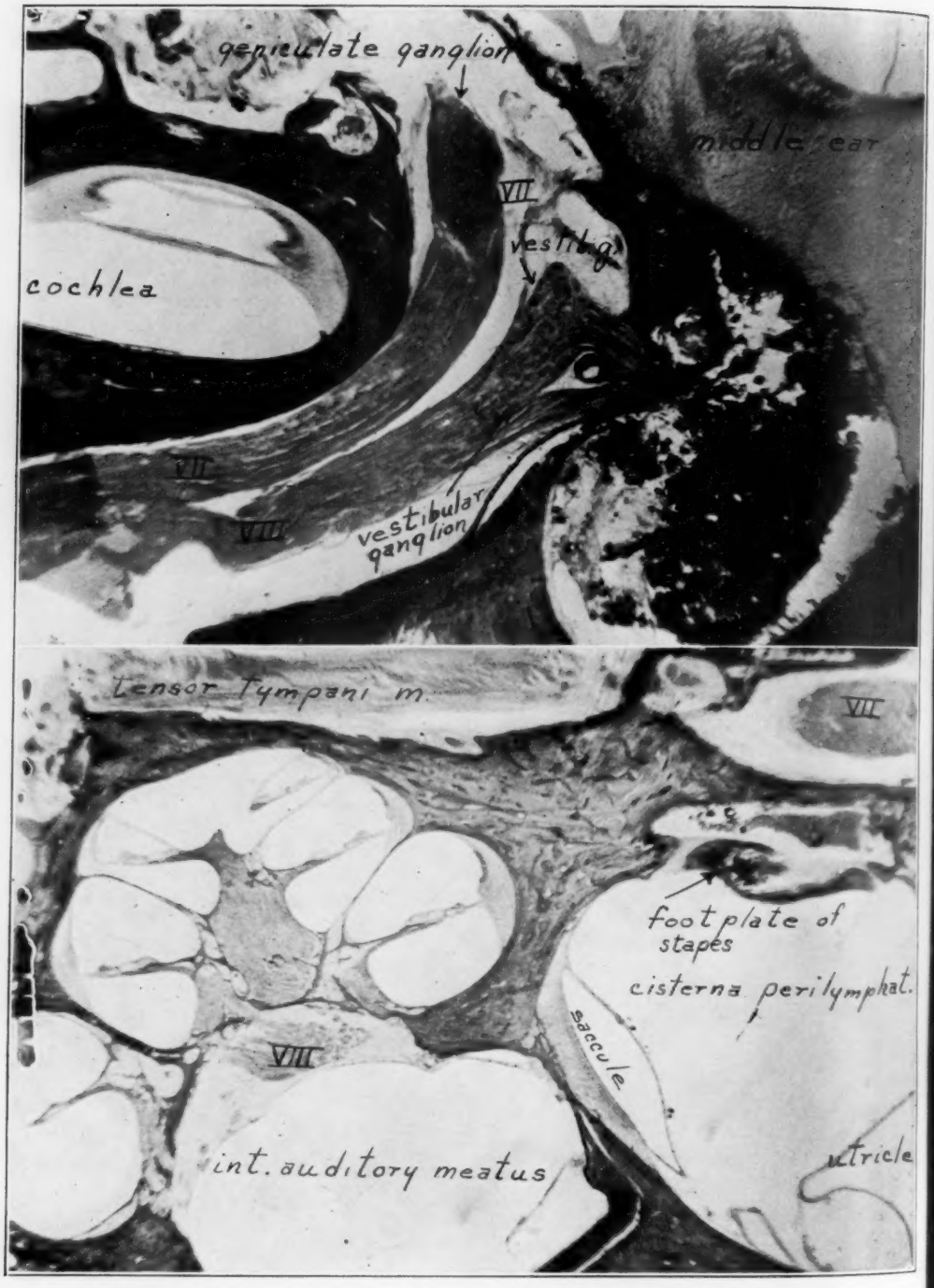

Fig. 4.—Complete destruction of the ampulla produced in the process of burnishing the walls of a fenestra with a silver amalgam. The cochlea is apparently normal. Experiment II; monkey 25, right ear; eight days after operation.

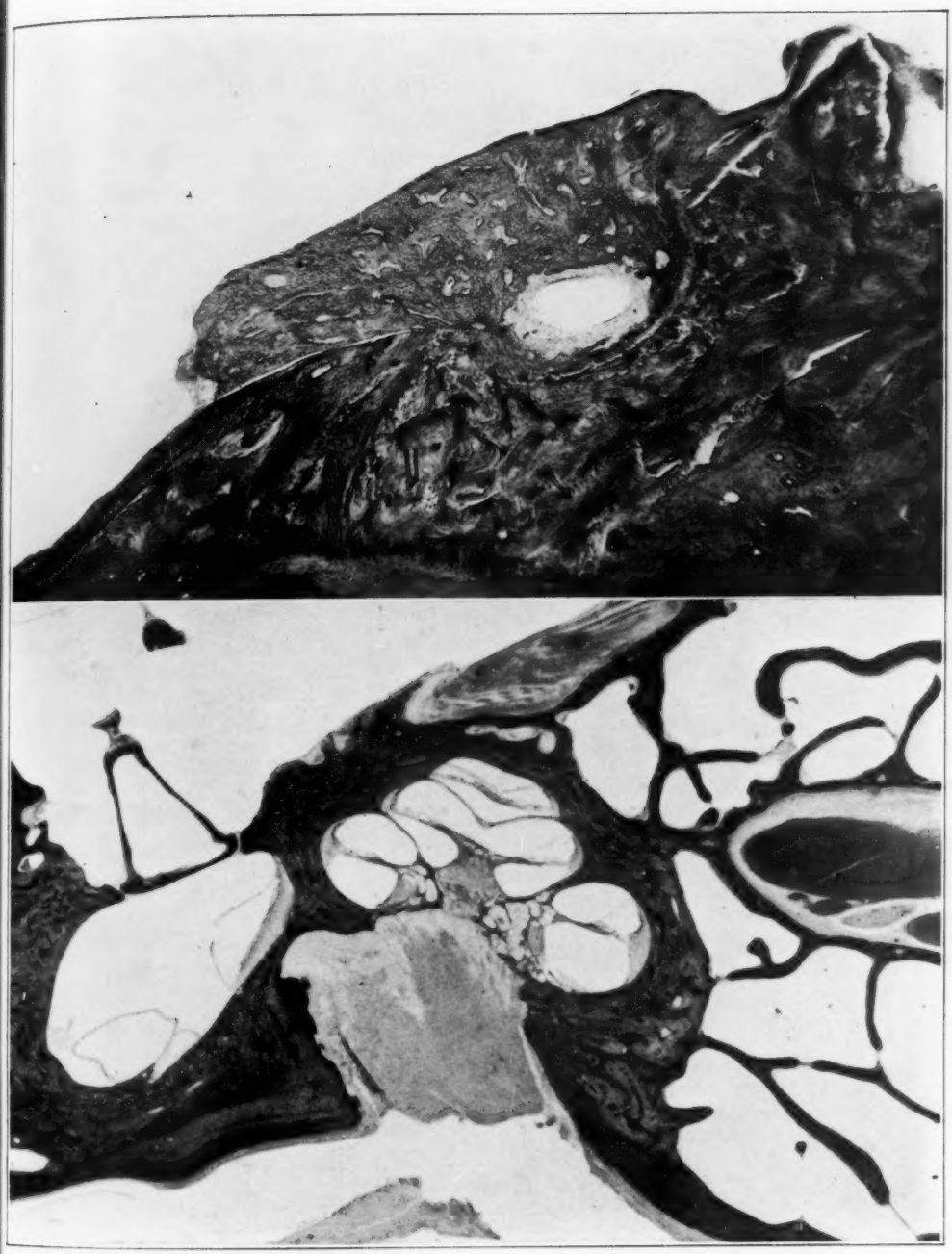

Fig. 5.—Obliteration of perilymphatic tissue by bone regenerating at the fenestra. The cochlea and the organ of Corti are apparently normal. Experiment II; monkey 29, left ear; seven months, five days after operation.

branous labyrinth does not result in degeneration of the remaining endolymphatic system.

Only in rare instances in which infection of the labyrinth followed such a surgical procedure did degeneration of the entire endolymphatic labyrinth and of the organ of Corti take place. Such infection will also travel and involve the cochlear nerve trunk.

Figures 6 and 7 show photomicrographs of the degeneration of the endolymphatic systems of the experimentally fenestrated labyrinths of the rhesus monkey caused by infection following partial destruction of the vestibular portion of the endolymphatic labyrinth.

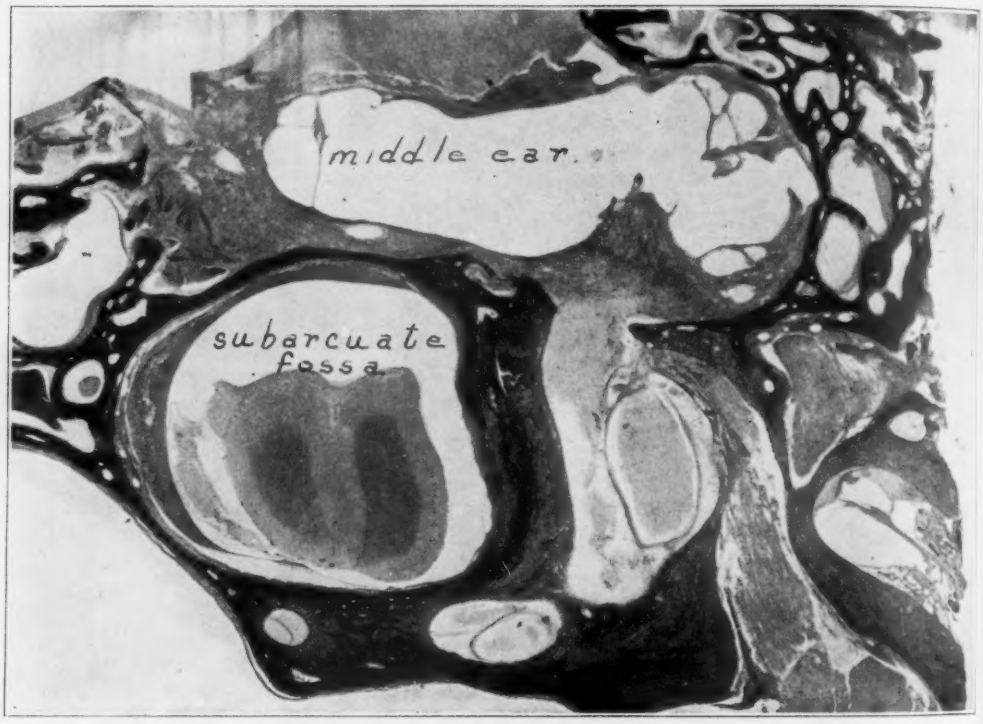

Fig. 6.—Otitis media with acute labyrinthitis in both vestibule and cochlea following fenestration. The organ of Corti shows degenerative changes. Experiment I; monkey 7, left ear; eight days after operation.

COMMENT

The aforementioned clinical and histologic observations led me to the following conclusions:

Partial destruction of the vestibular portion of the endolymphatic labyrinth is inadequate for the treatment of Ménière's disease. Since it does not as a rule result in degeneration of the cochlear part of the

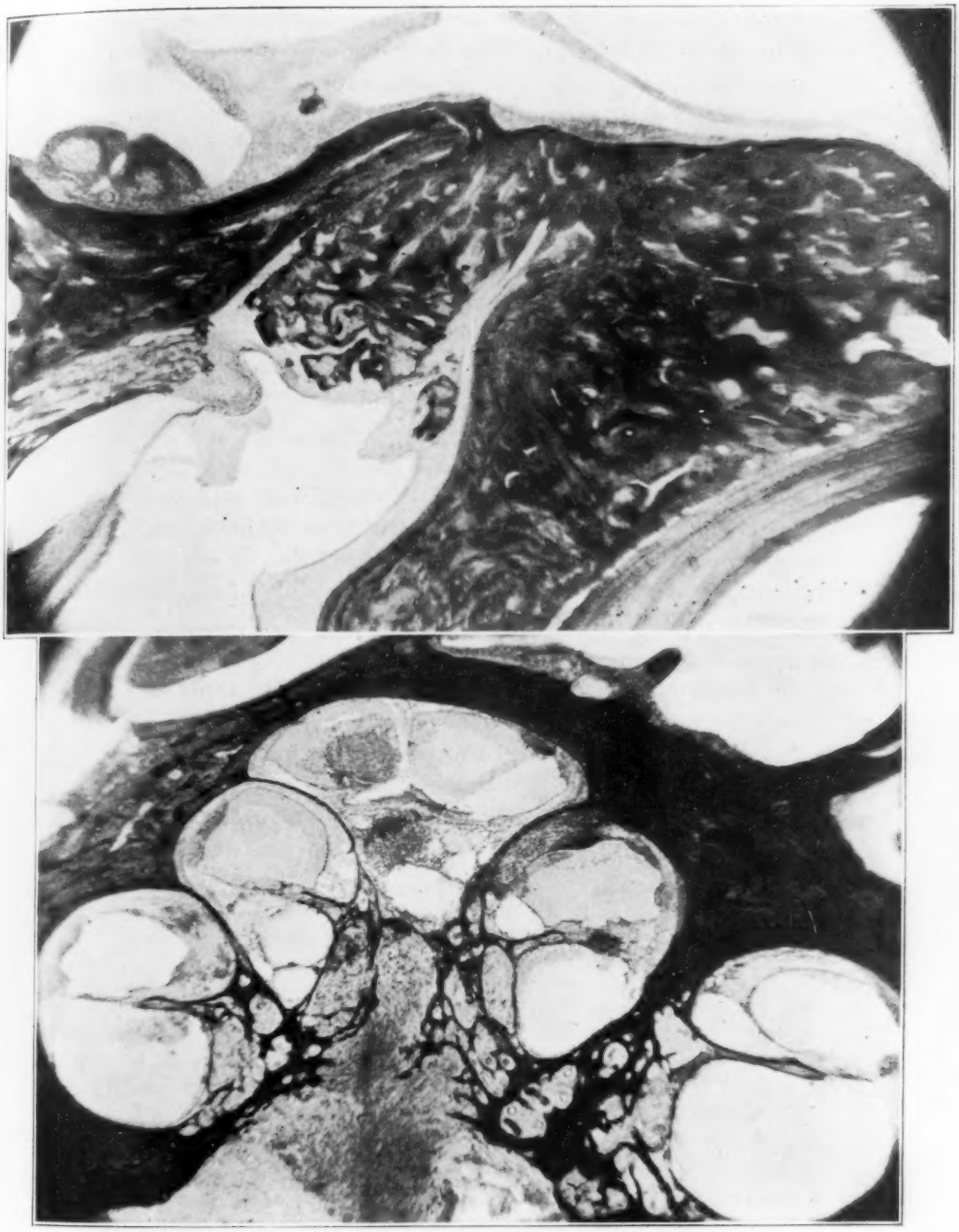

Fig. 7.—Acute purulent labyrinthitis following fenestration in which mature bone was tossed within the fenestra. Regeneration is complete, and osteoid degeneration of the mature bone may be seen near the crista. Reissner's membrane, the organ of Corti and the tectorial membrane have been destroyed. Experiment 11B; monkey 20, left ear.

endolymphatic labyrinth and of the organ of Corti, it cannot be expected to relieve the tinnitus. The destruction of the most sensitive and most readily activated external semicircular canal will prevent the sensation of vertigo but will not necessarily prevent the recurrence of endolymphatic hydrops. When on occasion the endolymphatic labyrinth and the organ of Corti do degenerate after the partial destruction of the vestibular part of the endolymphatic labyrinth, they do so only as a result of postoperative infection. When the endolymphatic labyrinth degenerates as a result of postoperative infection, the tinnitus is not relieved and, moreover, is perpetuated by the cochlear neuritis which results from the extension of the inflammation from the organ of Corti to the cochlear nerve trunk.

Total loss of hearing for both air-conducted and bone-conducted sound following partial labyrinthectomy in Ménière's disease is no proof that the endolymphatic labyrinth and the organ of Corti have completely degenerated. Destruction of the utricle is sufficient to prevent the remaining endolymphatic system from being mobilized by sound to the degree necessary for the stimulation of the preoperatively partially damaged organ of Corti, which may have remained unchanged after the surgical intervention.

It therefore became obvious that any technic expected to relieve both the vestibular and the cochlear symptoms must supply a reasonable basis for belief that the entire endolymphatic labyrinth will degenerate as a result thereof and thus render the recurrence of endolymphatic hydrops impossible. Furthermore, the degeneration of the endolymphatic labyrinth when accomplished through the technic employed must not be the result of excessive postoperative inflammation or infection.

TECHNIC FOR EFFECTING ATRAUMATIC AND ASEPTIC DEGENERATION OF THE ENTIRE ENDOLYMPHATIC SYSTEM

STEP 1. (*a*) *Creation of an Adequate Endaural Approach for Surgical Intervention Within the Tympanic Cavity* (fig. 8*A*).—Three incisions are made through the skin and periosteum lining the entire posterior wall of the external auditory canal. Incision 1 is begun at the inner margin of the superoposterior wall of the external auditory canal and carried outward along the superoposterior wall up to the apex of the membranous suprameatal triangle.

Incision 2 is begun at the inner margin of the inferoposterior wall of the external auditory canal and carried outward up to the lower end of the anterior border of the concha.

Incision 3 connects these two incisions in the posterior wall of the external auditory canal immediately adjacent to the posterior margin of the tympanic ring.

(*b*) *Subperiosteal Elevation and Eversion of the Skin and Periosteum Lining the Posterior Wall of the External Auditory Canal* (fig. 8*B* and *C*).—With a narrow subperiosteal elevator placed within the third incision so that it hugs the posterior

bony wall of the external auditory canal, the skin and periosteum lining the posterior wall of the canal are elevated from within outward up to the anterior border of the concha and everted backward over the margin of the concha.

With retractors inserted, the anterior margin of the outer mastoid cortex with spina Henle, the entire anterior mastoid cortex which forms the bony posterior wall of the external auditory canal, and the posterior aspect of the zygomatic process are exposed. The tympanic membrane is brought into view.

(*c*) *Widening of the External Auditory Canal* (fig. 9).—The spina Henle is removed. The entire superposterior, posterior and inferoposterior bony walls of the external auditory canal and the posteroinferior border of the zygomatic process are shaved and skeletonized with a sharp cutting, electrically driven steel burr. Care should be exercised not to open any of the mastoid cell structure.

STEP 2. *Endaural Exposure of the Incudostapedial Joint and Round Window Niche* (fig. 10).—(*a*) A narrow delicate periosteal elevator is placed against the skin-free innermost margin of the posterior bony canal wall, immediately adjacent to the posterior margin of the tympanic membrane. With the elevator hugging the bony margin and directed inward toward the tympanic cavity, the posterior margin of the annulus fibrosus of the tympanic membrane is dislodged from its position within the sulcus tympanicus.

(*b*) With the same instrument, the posterior half of the tympanic membrane is everted and placed at rest over the anterior half of the tympanic membrane. The incudostapedial joint and the round window niche are thus exposed to view.

STEP 3 (*a*) *Decompression of the Vestibular Part of the Labyrinth* (figs. 11, 12 and 13).—With a sharp 0000 Lempert curet, the tympanic ring in the region of the incudostapedial joint, which hides the crura of the stapes, is gently shaved to expose to direct view the entire crura of the stapes.

(*b*) With a sharp and narrow Lempert malleus clipper, the long process of the incus is cut near its attachment to the head of the stapedial crura; thus the stapes is freed from the incus.

(*c*) By engaging the crura of the stapes with a no. 11 or a no. 12 dental excavator and gently pulling the stapes in a direction anterior to and away from the oval window, one may easily remove the stapes with its crura and footplate intact. The vestibular perilymphatic space is thus widely opened, the perilymph drains out and the vestibular part of the endolymphatic labyrinth is exposed to the air of the tympanic cavity.

STEP 4. *Decompression of the Cochlear Part of the Labyrinth* (fig. 14).—With an electrically driven 1.5 mm. round polishing burr introduced into the round window niche, the membrane of the round window is removed and the round window proper enlarged The perilymphatic space of the scala tympani is thus widely opened, the perilymph drains out and the cochlear part of the endolymphatic labyrinth is exposed to the air of the tympanic cavity.

STEP 5. *Sealing of the Tympanic Cavity with Tympanic Membrane* (fig. 15).—The everted posterior half of the tympanic membrane is replaced in its original position. The dislodged posterior portion of the annulus fibrosus is fitted back into the sulcus tympanicus.

STEP 6. *Replacement of the Everted Membranous Lining of the Posterior Wall of the External Auditory Canal.*—(*a*) The retractors are removed, and the everted skin and periosteum are replaced in position to line the posterior bony wall of the external auditory canal.

(*b*) The canal is packed with paraffin gauze to hold the skin and periosteum in position. This packing is removed on the seventh postoperative day. The patient leaves the hospital on the tenth postoperative day. Three hundred units of

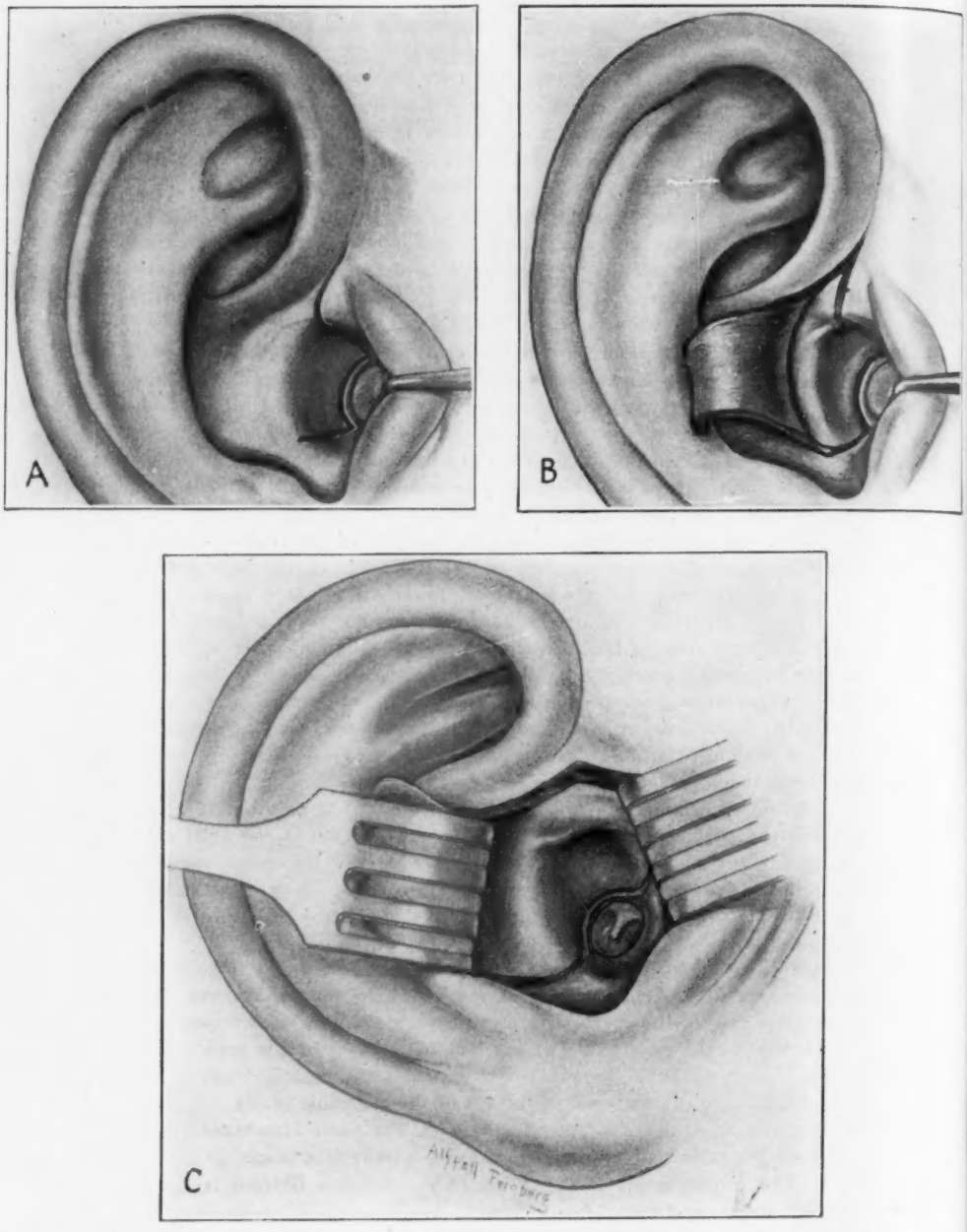

Fig. 8.—(*A*) Three endaural incisions are made through skin and periosteum. (*B*) The skin flap is subperiosteally freed from its bony attachments and everted outward over the anterior margin of the concha. (*C*) With retractors inserted, the outer mastoid cortex, the spina Henle, the entire anterior mastoid cortex and the tympanic membrane can now be seen.

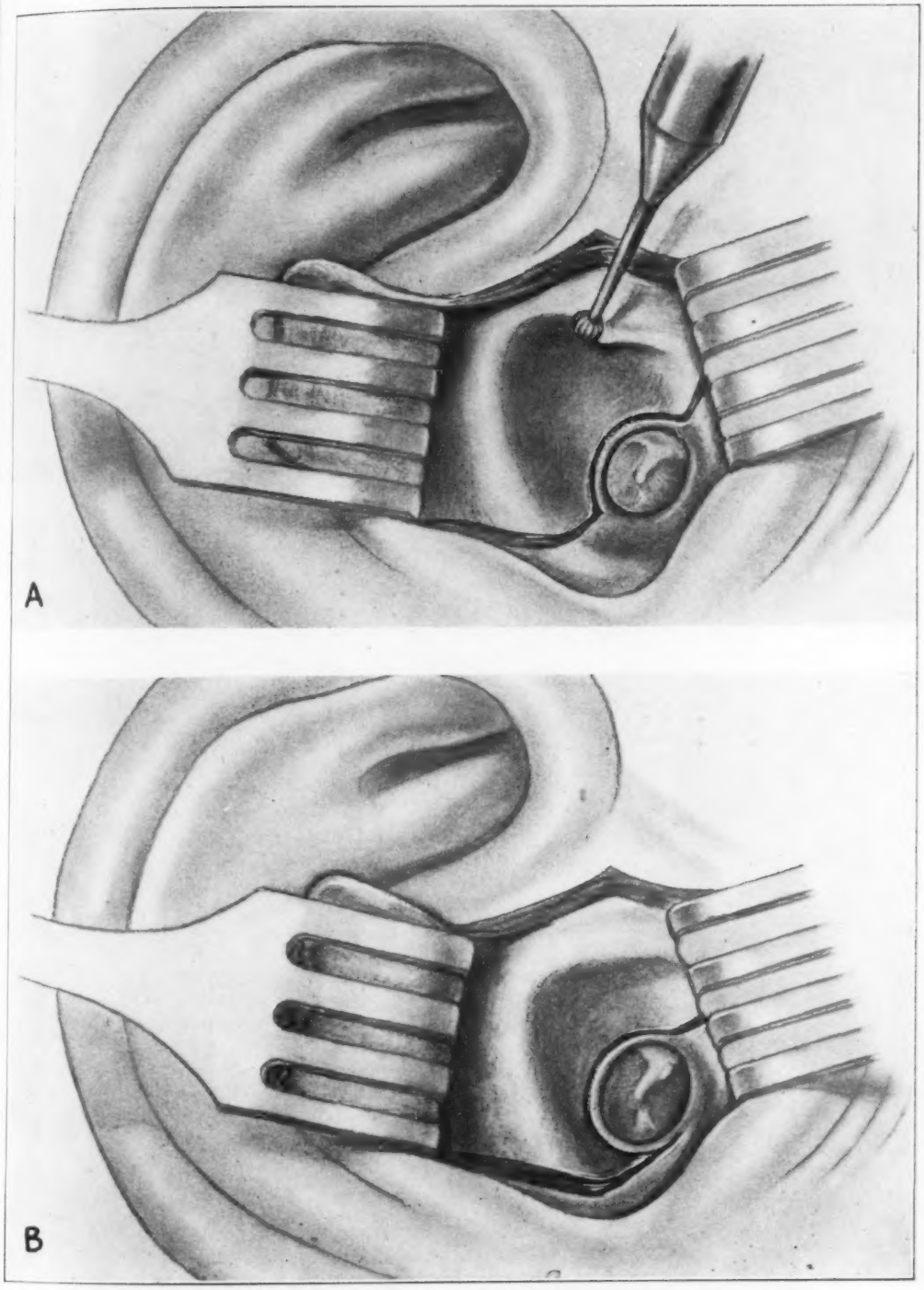

Fig. 9.—(*A*) With an electrically driven burr the spina Henle is removed and the external auditory canal is widened.

(*B*) The widening of the external auditory canal enhances the accessibility of the field of surgical intervention within the tympanic cavity.

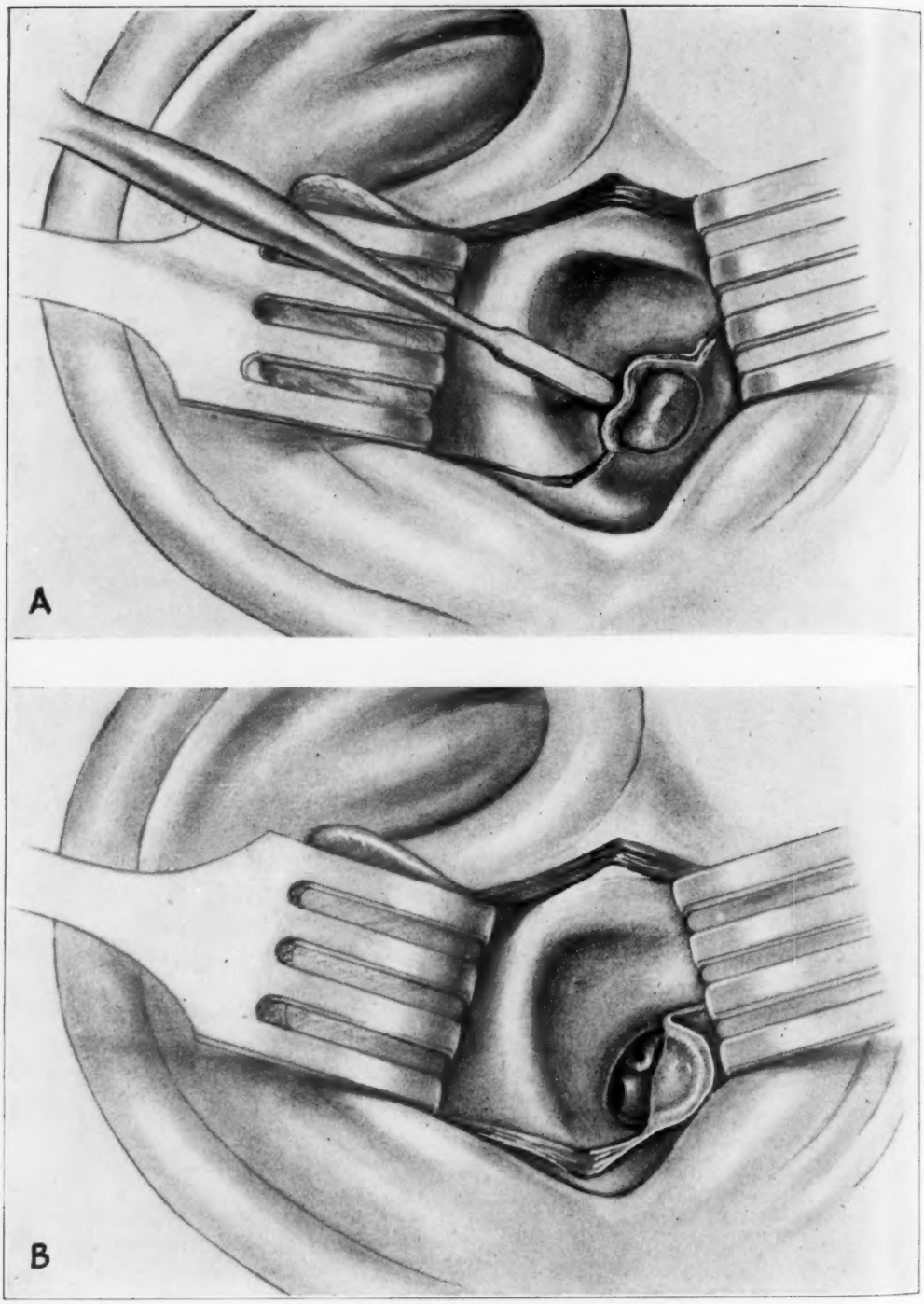

Fig. 10.—(*A*) The annulus fibrosus of the posterior half of the tympanic membrane is dislodged from the sulcus tympanicus with a narrow periosteal elevator.
(*B*) The dislodged portion of the tympanic membrane is everted, exposing to view the incudostapedial joint and the niche of the round window.

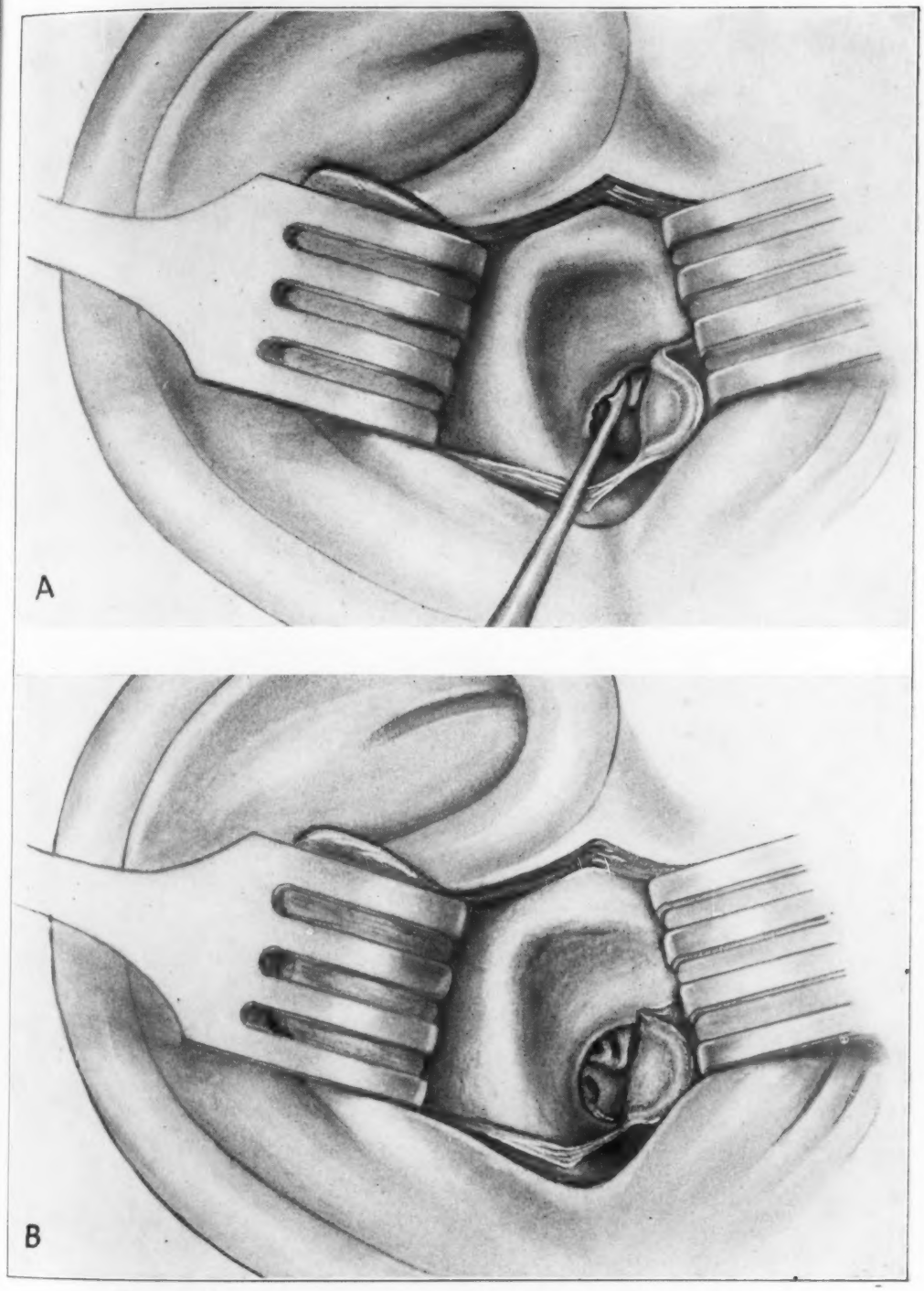

Fig. 11.—(*A*) To expose the crura of the stapes, the portion of the bony annulus tympanicus which hides it is removed.
(*B*) The stapedial crura can now be seen.

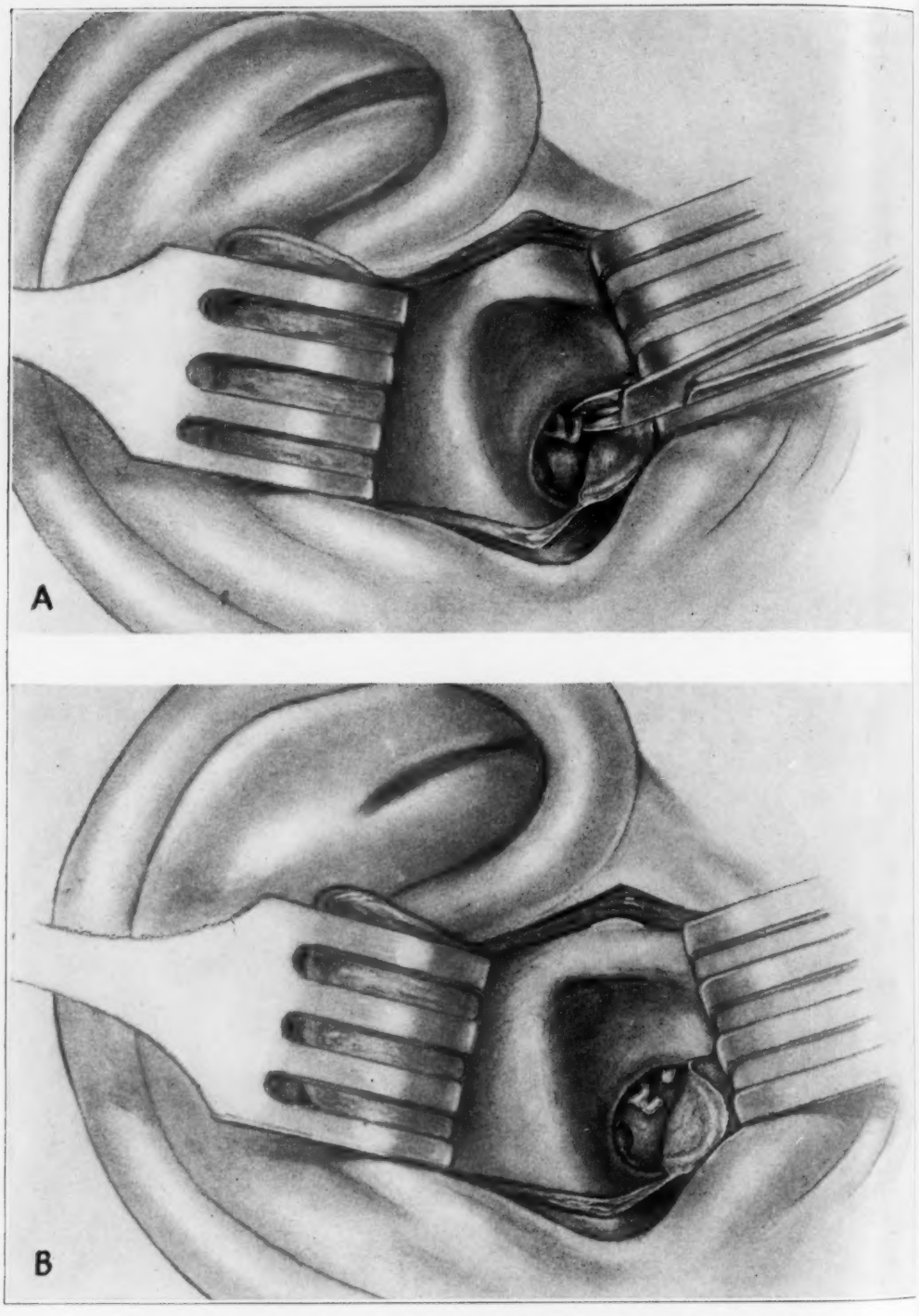

Fig. 12.—(*A*) With a sharp, narrow steel clipper the long process of the incus is bisected close to its attachment to the head of the stapes.
(*B*) The bisected long process of the incus is seen.

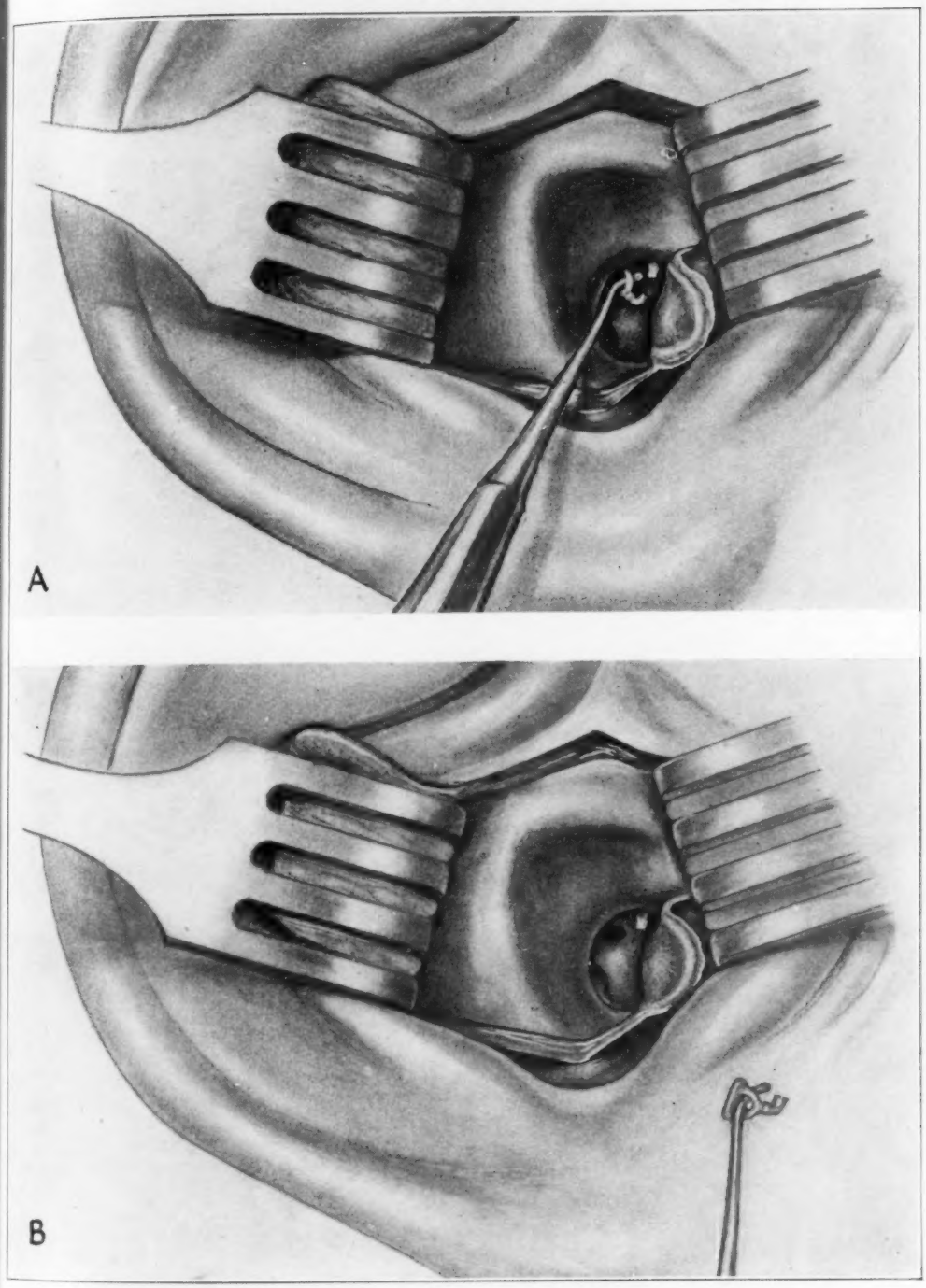

Fig. 13.—(*A*) The vestibular part of the labyrinth is decompressed by hooking and removing the stapes with its foot plate from the oval window
(*B*) The disengaged stapes and the decompressed vestibule are seen.

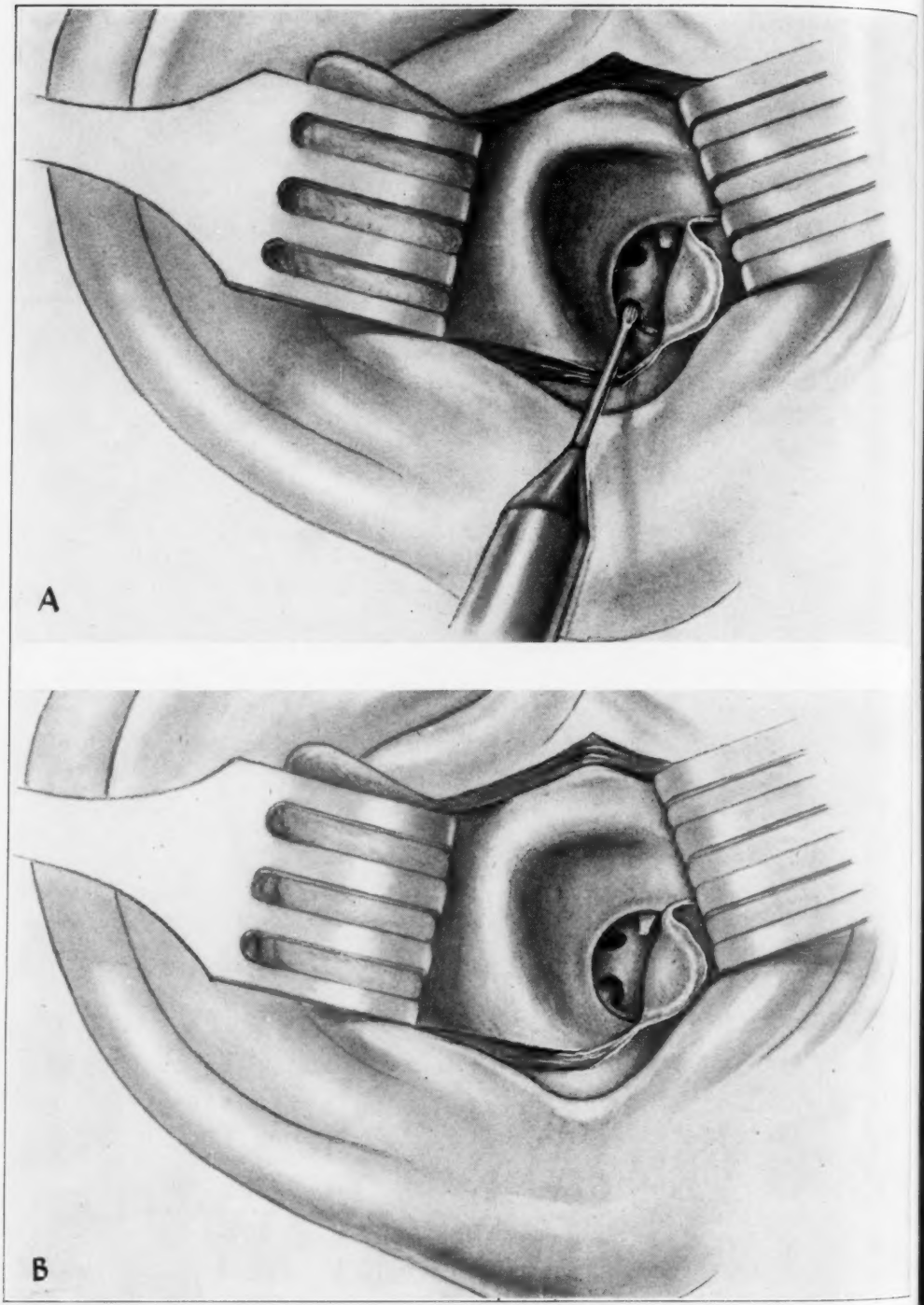

Fig. 14.—(*A*) The oval window is seen, with the stapes removed. The cochlear part of the labyrinth is now decompressed by inserting an electrically driven 2 mm. polishing burr into the round window niche and destroying the membrane of the round window.

(*B*) The decompressed vestibule and cochlea are now seen.

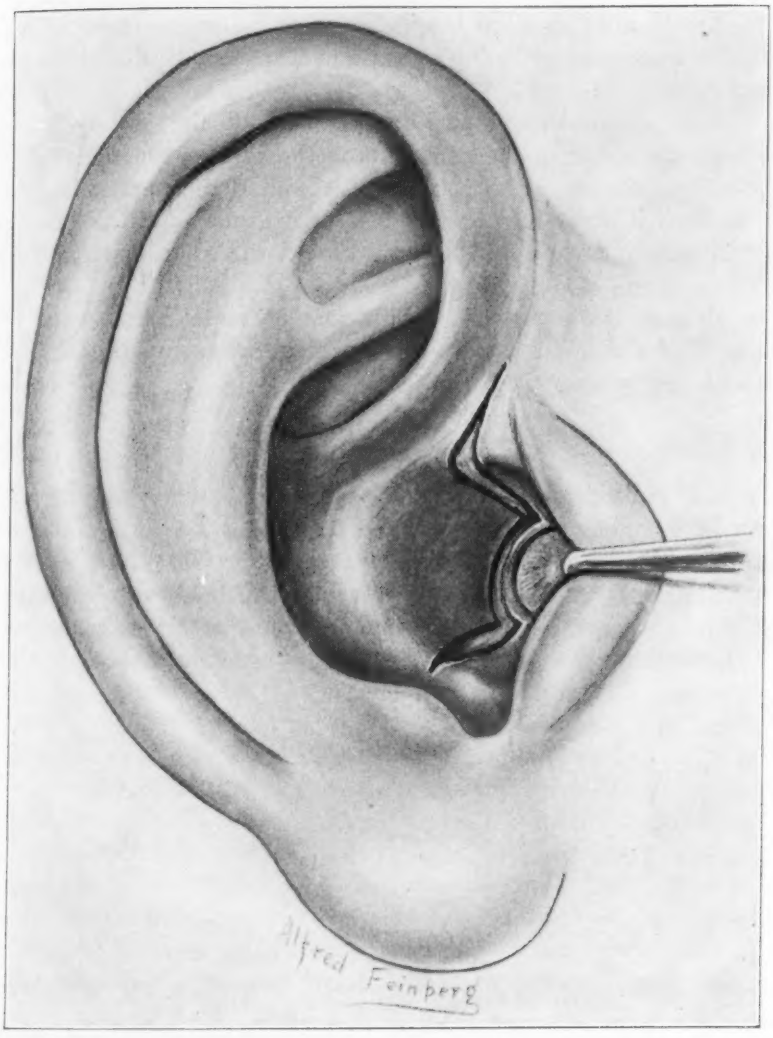

Fig. 15.—The dislodged portion of the tympanic membrane and the temporarily everted skin and periosteum of the external auditory canal are replaced to their original positions.

penicillin is given daily during the entire hospital stay to reduce postoperative inflammation to an absolute minimum and to prevent postoperative infection.

ADVANTAGES

1. The removal of the stapes and the round window membrane without traumatizing either the temporal bone or the endolymphatic labyrinth when executed under sterile precautions with the administration of penicillin results in aseptic degeneration of the entire endolymphatic labyrinth and of the organ of Corti.

2. By effecting complete degeneration of the endolymphatic labyrinth the recurrence of endolymphatic hydrops can be definitely prevented.

3. Such atraumatic aseptic degeneration of the endolymphatic labyrinth can result in the cure of both the vertigo and the tinnitus in Ménière's disease. Of the 10 patients who were operated on with this technic, 9 were freed from both vertigo and tinnitus. One was freed of vertigo but not of tinnitus, though the intensity of the tinnitus even in this case has greatly diminished.

4. Because the postoperative inflammatory reaction following this technic is of a minimal degree, the convalescence is much more rapid and attended by much less discomfort.

SUMMARY

A new technic for effecting atraumatic and aseptic degeneration of the endolymphatic labyrinth for the cure of both vertigo and tinnitus in Ménière's disease is described.

The clinical and histologic observations which have indicated the necessity for the development of such a technic are given.

The advantages which this technic offers are stated.

THE BACTERIAL FLORA OF THE NASOPHARYNX IN RELATION TO THE COMMON COLD

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AND

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IT IS NOW the consensus that the primary cause of the infectious common cold is a filterable virus. This initial viral infection, however, is usually followed by secondary bacterial infections, of varying severity, which are generally regarded as being largely responsible for such complications of the disease as paranasal or accessory sinusitis, pharyngitis, laryngitis, tracheobronchitis, pneumonia and secondary infections of the lesions of pulmonary tuberculosis. Just how or why the primary viral infection predisposes to these important secondary bacterial infections is unknown, but it is commonly thought that the local resistance of the tissues is reduced by the virus. On the other hand, the concomitance of virus and different bacteria may be due to symbiotic infection, or it may be the result of an increase of the virulence of various streptococci, staphylococci, pneumococci and other micro-organisms commonly occurring in the upper respiratory tract or gaining access to it.

Be that as it may, the infectious common cold and the secondary bacterial infections constitute a disease of such economic and other importance that the causation, the prophylaxis and the treatment of this disease properly warrant deep interest and investigation. Needless to state, there is no hope of evolving a method of successfully immunizing or vaccinating against the disease until the virus has been cultivated for the preparation of vaccine, and this, unfortunately, has not yet been accomplished. Whether or not, however, autogenous and

This investigation was aided in part by the William S. Merrell Company, Cincinnati.

From the Department of Bacteriology and Immunology of Temple University School of Medicine and the Research Institute of Cutaneous Medicine.

stock bacterial vaccines are of value in immunizing against the secondary bacterial infections is a subject of importance concerning which there continues to be a difference of opinion. Furthermore, the presently known sulfonamide and antibiotic compounds are ineffective against the virus, although some are effective in the treatment of the secondary bacterial infections, with special reference to sulfadiazine, sulfamerazine and penicillin.

PURPOSE OF INVESTIGATION

The purpose of our investigation was (1) to compare the nasopharyngeal bacterial flora of normal adults with that of adults who were suffering from the common cold, particularly the early and late stages of the disease; (2) to determine the relative incidence and quantitative importance of different micro-organisms in the nasopharyngeal cultures of those who showed secondary bacterial infections; (3) to determine the influence of season on the bacterial flora of the nasopharynx in normal persons and (4) to ascertain whether or not particular micro-organisms are responsible for the secondary infections. Needless to state, it is quite likely that the bacterial flora of normal adults, as well as that of adults subject to the common cold, may vary according to geographic factors, but it is likely that the results of this investigation may be generally applicable to the more northern localities of the United States at least.

TECHNIC EMPLOYED

With but few exceptions, all the normal subjects investigated and those with the common infectious cold were members of our staffs, medical students, laboratory technicians and nurses. Due care was exercised in excluding as far as possible persons with any chronic infection of the nasal accessory sinuses, including those with its complications, such as nasopharyngitis or laryngitis. Nasopharyngeal material was taken from both groups, and cultured, between November 1946 and May 1947, inclusive. During that period 1,132 cultures were made and studied, embracing 332 cultures of material taken from 209 normal adults and 800 cultures of material taken from 146 adults who had a total of 209 attacks of the common cold during the seven month period of investigation.

In isolating micro-organisms from the upper nasopharynx, material was collected with bent sterile swabs (prepared of stiff nicochrome wire) without permitting it to become contaminated with any contents of the oral cavity. These swabs were placed in small tubes of sterile broth until plate cultures could be made; then two plates of tryptose blood agar (10 per cent sterile horse blood) were streaked in such manner as to permit the growth of isolated colonies readily adapted for identification. To aid in the identification of colonies, a small paper disk saturated with a solution of penicillin (15 units per cubic centimeter) was placed on each plate according to the technic of Bondi, Spaulding, Smith and Dietz[1]; this

1. Bondi, A., Jr.; Spaulding, E. H.; Smith, D. E., and Dietz, C. C.: A Routine Method for the Rapid Determination of Susceptibility to Penicillin and Other Antibiotics, Am. J. M. Sc. **213**:221-226 (Feb.) 1947.

procedure was particularly useful as an aid in the identification of Hemophilus influenzae and certain other micro-organisms as well. One plate was incubated aerobically and the second anaerobically when the different bacteria were to be identified.

RESULTS OBSERVED

The micro-organisms found in cultures of the nasopharyngeal material taken from 198 normal controls and in 209 cases of the common cold are summarized in table 1 after a method similar to that employed by Julianelle and Siegel.[2] It will be observed that the same micro-organisms were isolated from the adults with the common cold

TABLE 1.—*Incidence of Each Variety of Micro-Organisms in Nasopharynxes of Normal Adults and Adults with the Common Cold*

Variety	Percentage from Which the Given Variety Was Isolated*	
	Normal Controls	Cases of the Common Cold
Streptococcus, alpha	23.4	22.7
Streptococcus, beta (group A)	1.8	4.0
Streptococcus, beta (other types)	20.1	18.8
Streptococcus, gamma	13.6	13.8
Staphylococcus albus	3.1	3.7
Staphylococcus aureus	3.8	5.8
Hemophilus influenzae	0.4	0.7
Neisseriae	24.8	22.3
Pneumococci	3.5	3.2
Diphtheroid bacilli	4.0	3.4
Miscellaneous varieties†	1.4	1.1

*The percentage is based on 198 normal controls and 209 cases of the common cold.
†These included Pseudomonas pyocyanea (Ps. aeruginosa), Klebsiella pneumoniae and yeasts.

as from the normal adults; in no instance was an unusual bacterium found in the cultures of the persons suffering from the common cold. In these persons, however, the incidence of beta hemolytic streptococci of group A and staphylococci was slightly but definitely higher than in the normal controls, along with a slightly increased incidence of H. influenzae. At the same time it will be observed that the incidence of the Neisseriae, with special reference to N. catarrhalis, was slightly lower in patients with the common cold than in normal controls, an observation which indicates that Neisseria infection may not be as important in the common cold as is generally surmised. Furthermore, the incidence of pneumococci was essentially the same in the naso-

2. Julianelle, L. A., and Siegel, M.: The Epidemiology of Acute Respiratory Infections Conditioned by Sulfonamides: II. Gross Alterations in the Nasopharyngeal Flora Associated with Treatment, Ann. Int. Med. **22**:10-20 (Jan.) 1945.

pharyngeal cultures of both series of persons although, as shortly to be discussed, multiple cultures of patients with the common cold have shown a definite increase of pneumococci as compared with multiple cultures of normal controls.

These comparative differences are shown more definitely in table 2, in which the percentage of the isolations of each of the various micro-organisms is based on 332 cultures of 198 normal controls and 800 cultures obtained in 209 cases of the common cold. For some persons, as many as 10 to 15 cultures were made. Consequently, the data summarized in this manner must be broadly interpreted, because

TABLE 2.—*Incidence of Each Variety of Bacteria in Nasopharyngeal Cultures of Normal Adults and Adults with the Common Cold*

Variety	Percentage of Cultures Showing Given Variety	
	Normal Controls*	Cases of the Common Cold†
Streptococcus, alpha	84.3	83.9
Streptococcus, beta (group A)	6.6	14.8
Streptococcus, beta (other types)	72.3	69.4
Streptococcus, gamma	49.1	51.0
Staphylococcus albus	11.1	13.5
Staphylococcus aureus	13.6	21.5
Hemophilus influenzae	1.5	2.8
Neisseriae	89.5	82.4
Pneumococci	12.7	11.9
Diphtheroid bacilli	14.5	12.6
Miscellaneous varieties‡	5.1	4.1

*The percentage is based on 332 cultures of nasopharyngeal material taken from 198 normal controls.

†The percentage is based on 800 cultures of nasopharyngeal material obtained in 209 cases of the common cold.

‡These include Pseudomonas pyocyanea, Klebsiella pneumoniae and yeasts.

the percentage incidence of any particular micro-organism occurring in all cultures of a given person would be increased according to the number of cultures made and examined. To determine whether or not this is the case, further data were assembled in table 3, in which the percentage of isolations is based on the "average culture" of each person. This average culture represented the various bacteria isolated in 50 per cent or more of all the cultures made on a given person. The incidence of the various micro-organisms appearing in 94 average or representative cultures of 94 persons each of whom had a single cold is shown. For comparison, data are summarized with regard to a group of normal controls in whose interest multiple cultures had been made. The results tabulated in this manner, however, have like-

wise shown an increase of beta hemolytic streptococci of group A, Staphylococcus aureus and pneumoccoci in cases of the common cold.

As shown in table 4, the percentage of subjects whose cultures showed coagulase-positive staphylococci was practically the same in the group with the common cold (54.6 per cent) as in the group of normal controls (54.9 per cent).

TABLE 3.—*Average Percentage of Isolations of Each Variety of Bacteria Found in Nasopharyngeal Cultures of Normal Adults and Adults with the Common Cold*

Variety	Average Percentage of Isolations	
	Normal Adults*	Adults with the Common Cold†
Streptococcus, alpha	22.9	23.1
Streptococcus, beta (group A)	1.0	3.3
Streptococcus, beta (other types)	22.4	20.6
Streptococcus, gamma	15.7	15.1
Staphylococcus albus	4.3	1.6
Staphylococcus aureus	3.8	4.9
Hemophilus influenzae	1.0	0.5
Neisseriae	23.8	24.5
Pneumococci	2.9	3.3
Diphtheroid bacilli	2.4	3.0

*The percentage is based on the "average culture" of 50 normal controls (average of 3.9 cultures per person).
†The percentage is the "average culture" of 94 adults with the common cold (average of 7.7 cultures per person).

TABLE 4.—*Coagulase Activity of Staphylococci Isolated, in Nasopharyngeal Cultures, from Normal Adults and Adults with the Common Cold*

Group	Number	Percentage Whose Staphylococci Were Coagulase Positive	Percentage Whose Staphylococci Were Coagulase Negative
Normal adults	51	54.9	45.1
Adults with colds	185	54.6	45.4

The specific serologic types of the pneumococci isolated in the 28 cultures of pneumococci of the 198 normal controls and in the 41 cultures of pneumococci obtained in 209 cases of the common cold were also determined, the results being summarized in table 5. In the cultures representing the normal controls, 13 different types were identified, with 2 cultures in which the type or types could not be determined. In the cultures representing cases of the common cold, 15 different types were identified, with 2 cultures in which the type or types could be determined. It will be observed that types I and II

were conspicuous by their absence; type 1 was found only once in the cultures made in cases of common cold, while types III, VI, X, XI and XIX occurred most frequently, especially type VI.

TABLE 5.—*Incidence of Each Specific Type of Pneumococci in Nasopharyngeal Cultures of Normal Adults and Adults with the Common Cold*

Group	Number of Cultures of Pneumococci	NUMBER OF CULTURES SHOWING GIVEN TYPE OF PNEUMOCOCCI																		
		I	III	VI	VII	VIII	IX	X	XI	XIV	XV	XVIII	XIX	XX	XXII	XXIII	XXIV	XXVIII	XXXII	*
Normal adults	28	0	4	5	0	0	1	1	1	0	1	1	2	1	0	2	3	3	1	2
Adults with cold	41	1	4	8	2	1	1	4	4	1	0	0	5	0	1	2	1	3	1	2

*The type could not be identified.

TABLE 6.—*Quantitative Estimation of the Growth of Each Variety of Micro-Organisms Found in Nasopharyngeal Cultures of Normal Adults and Adults with the Common Cold**

Variety	Percentage of Cultures Showing Given Amount of Growth†							
	Normal Controls				Cases of the Common Cold			
	+	++	+++	++++	+	++	+++	++++
Streptococcus, alpha	9	35	37	19	11	31	33	25
Streptococcus, beta (group A)	55	9	23	14	55	23	13	9
Streptococcus, beta (other types)	39	34	20	7	47	29	15	9
Streptococcus, gamma	47	40	10	3	46	44	9	1
Staphylococcus albus	57	19	13	11	53	25	6	16
Staphylococcus aureus	49	18	15	18	51	27	11	11
Hemophilus influenzae	20	60	20	0	9	27	32	32
Neisseriae	17	37	28	18	14	34	31	21
Pneumococci	17	30	17	36	17	26	28.5	28.5
Diphtheroid bacilli	73	23	4	0	65	30	5	0
Miscellaneous varieties‡	53	6	23	18	34	34	23	9

*The total numbers on which this table is based are 198 normal adults (332 cultures) and 209 cases of the common cold (800 cultures).
†The amount of growth is indicated as follows: + (occasional colony), ++ (few colonies), +++ (moderate number of colonies) and ++++ (many colonies).
‡These include Pseudomonas pyocyanea, Klebsiella pneumoniae and yeasts.

An attempt was made to measure quantitatively the number of colonies of the different micro-organisms on the plates of tryptose blood agar. The results, based on 332 cultures of 198 normal controls and 800 cultures made in 209 cases of common cold, are summarized

in table 6, in which the percentages of cultures of each of various micro-organisms showing 1,2,3, and 4 plus growths, respectively, are shown. It will be observed that the beta hemolytic streptococci, staphylococci, pneumococci and H. influenzae were numerically more numerous in cultures in the cases of the common cold than in cultures of normal controls.

Of additional interest are the results of 89 cultures made in 39 cases of the common cold during the early stage (first four days) and 132 cultures made in the same cases during the late (after the fourth day) stage of the disease. These results are summarized in table 7, in which it will be observed that the percentages of positive cultures of

TABLE 7.—*Incidence of Each Variety of Bacteria in Nasopharyngeal Cultures Which Were Made in 39 Cases of the Common Cold Taken During the Early and the Late Stage of the Disease*

Variety	Percentage Incidence	
	Early Stage*	Late Stage†
Streptococcus, alpha	87.6	86.4
Streptococcus, beta (group A)	14.6	15.2
Streptococcus, beta (other types)	62.9	68.2
Streptococcus, gamma	56.2	56.8
Staphylococcus albus	9.0	12.1
Staphylococcus aureus	16.9	17.4
Hemophilus influenzae	2.2	2.3
Neisseriae	79.8	80.3
Pneumococci	7.9	11.4
Diphtheroid bacilli	13.5	11.4

*The incidence is based on 89 cultures made during the first four days.
†The percentage is based on 132 cultures made after the fourth day.

beta hemolytic streptococci, staphylococci and pneumococci were somewhat higher during the late than during the early stage of the common cold. On the quantitative basis, beta hemolytic streptococci (especially group A), Staph. aureus and pneumococci were also more numerous during the late than during the early stage of the disease, as shown in table 8.

As expected, seasons have had some influence on the bacterial flora of the nasopharynx in normal persons in Philadelphia. As shown in table 9, based on the results of single cultures of material taken from 204 adults, gamma streptococci, staphylococci, pneumococci, H. influenzae and diphtheroid bacilli occurred somewhat more frequently during the autumn and winter months (November to March, inclusive) than during the spring months (April and May). Curiously

enough, however, beta hemolytic streptococci (including group A) and the Neisseriae were found somewhat more frequently during April and May.

TABLE 8.—*Quantitative Estimation of the Growth of Each Variety of Organisms Present in Nasopharyngeal Cultures in 39 Cases of the Common Cold Taken During the Early and the Late Stage of the Disease**

Organisms	Percentage of Cultures Showing Given Amount of Growth†							
	Early Stage (89 Cultures)				Late Stage (132 Cultures)			
	+	++	+++	++++	+	++	+++	++++
Streptococcus, alpha	8	28	32	32	11	27	44	18
Streptococcus, beta (group A)	46	39	15	0	45	30	20	5
Streptococcus, beta (other types)	48	27	9	16	52	27	12	9
Streptococcus, gamma	34	56	8	2	51	40	9	0
Staphylococcus albus	75	12.5	0	12.5	63	31	0	6
Staphylococcus aureus	67	20	6.5	6.5	35	39	13	13
Neisseriae	17	24	29.5	29.5	14	35	26	25
Pneumococci	14	43	14	29	13	7	47	33
Diphtheroid bacilli	75	17	8	0	40	53	7	0

*The early stage is that observed during the first four days; the late stage, that after the first four days.
†The amount of growth is indicated as follows: + (occasional colony); ++ (few colonies); +++ (moderate number of colonies); ++++ (many colonies).

TABLE 9.—*Seasonal Influence on the Incidence of Each Variety of Bacteria Observed in Nasopharyngeal Cultures of Normal Adults*

Variety of Organisms	Percentage of Positive Cultures in Given Period	
	November—March*	April—May†
Streptococcus, alpha	23.7	24.1
Streptococcus, beta (group A)	1.4	2.7
Streptococcus, beta (other types)	18.1	22.0
Streptococcus, gamma	15.8	12.3
Staphylococcus albus	3.4	2.7
Staphylococcus aureus	4.5	2.4
Hemophilus influenzae	1.1	0
Neisseriae	22.0	26.3
Pneumococci	3.1	2.7
Diphtheroid bacilli	5.4	4.3
Miscellaneous varieties‡	1.4	0.5

*The percentage is based on the results of 100 cultures of nasopharyngeal material taken from 100 adults.
†The percentage is based on the results of 104 cultures of nasopharyngeal material taken from 104 adults.
‡These include Pseudomonas pyocyanea, Klebsiella pneumoniae and yeasts.

COMMENT

The results of this investigation indicate, therefore, that beta hemolytic streptococci of group A, staphylococci and pneumococci are important in the causation of the secondary bacterial infections associated with the common cold. Of these, beta hemolytic streptococci of group A and staphylococci appear to be of most importance. But while a large percentage of cases of pneumococcic pneumonia are known to follow antecedent infections of the upper respiratory tract, including the common cold, and while 50 to 60 per cent of cases are due to infections with pneumococci of types I and II, yet these two types were conspicuous by their absence in this investigation. Under the circumstances it appears that the role played by the common cold in the causation of pneumonia of types I and II, at least, may be largely that of reducing the local resistance of the lower respiratory tract and thus favoring infection with pneumococci that gain access to the tract through the patient's coming in contact with carriers.

It is not our purpose at the present time, however, to discuss the manner in which the results observed relate to methods of immunizing or vaccinating against the secondary bacterial infections of the common cold. In this connection it may be briefly stated, however, that Kolmer[3] has found that the parenteral administration of carefully prepared vaccines has apparently proved of prophylactic value in a large percentage of persons who do not have pronounced chronic infection of the nasal accessory sinuses and especially chronic ethmosphenoidal infection. In chronic sinusitis acute exacerbations or relapses of the disease are commonly and erroneously regarded as attacks of the infectious common cold. If vaccines are employed for active immunization, it appears that they should be prepared of beta hemolytic and gamma streptococci, Staph. aureus and H. influenzae with special reference to beta hemolytic streptococci of group A. Obviously, it is impossible to include adequate numbers of all the specific types of pneumococci, but, if any are included, it would appear advisable to employ types III, VI, X, XI and XIX so far as the results of this investigation are concerned.

SUMMARY AND CONCLUSIONS

Various micro-organisms were identified in 1,132 cultures of material swabbed from the upper nasopharynxes of 209 normal adults (332 cultures) and 146 adults who had a total of 209 attacks of the common cold (800 cultures) during the period of investigation.

3. Kolmer, J. A.: Chemotherapy and Biotherapy: Their Relation to the Prevention and Treatment of Diseases of the Ear, Nose and Throat, Arch. Otolaryng. **40**:17-28 (July) 1944.

All micro-organisms isolated from adults suffering with the common cold were likewise isolated from normal adults.

Beta hemolytic streptococci of group A, staphylococci and pneumococci, however, occurred more frequently and in larger numbers in the cultures made in the cases of the common cold than in the cultures of material taken from the normal controls; apparently these micro-organisms are important in the causation of the secondary bacterial infections associated with the common cold and its complications.

Beta hemolytic streptococci of group A, staphylococci and pneumococci also occurred more frequently and in larger numbers in the late than in the early stages of the common cold.

Gamma streptococci, staphylococci, pneumococci, H. influenzae and diphtheroid bacilli occurred more frequently in cultures of material from normal adults during the autumn and winter months than during the spring months; beta hemolytic streptococci and the Neisseriae occurred more frequently during the spring than during the winter months.

The incidence of coagulase-positive staphylococci was practically the same in cultures made in cases of the common cold as in cultures of material from normal controls.

Thirteen different types of pneumococci were observed in cultures of material from normal controls and fifteen different types in cultures made in cases of the common cold. In cultures representing cases of the common cold, types III, VI, X, XI and XIX occurred most frequently, especially type VI.

These results are briefly discussed in relation to the possibility of actively immunizing persons against the secondary bacterial infections of the common cold.

CHOLESTEATOMA AND CHRONIC OTITIS

Etiologic Problems

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THE TERM "CHOLESTEATOMA" dates from the beginning of the nineteenth century, created by the anatomist J. Miller, with the implication that the lesion should be regarded as a genuine tumor. According to the pathologists Cruveilhier, Rokitansky and Virchow, cholesteatoma often occurs in the ear but rarely at other places in the skull—for example, in the pia mater or the os petrosum. They did not, however, discard the view that cholesteatoma, even when occurring in the ear, is a genuine tumor. The otologists Gruber (1862) and von Troeltsch (1868) showed that in clinical treatment chronic otitis is frequently found in conjunction with cholesteatoma. They contended that cholesteatoma of the ear—in distinction from that of the pia mater—is not a real tumor and that it is caused by chronic otitis. Brock considered this view to be *naheliegend und einleuchtend* (reasonable and enlightening). It seems to be generally accepted to this day.[1]

In clinical practice one often finds oneself confronted with recurring combinations of two or more pathologic phenomena, each of which may manifest itself independently. In certain cases the coexistence may be quite fortuitous; in others it may be due to a real connection. The phenomena may be connected simply as parallel effects of a common cause, or they may be really related as cause and effect. In the latter case it remains to be determined which is the cause and which the effect.

When one is faced with such a recurring combination, the tendency is to maintain that there is, that there must be, some causal connection. Unfortunately, one is also prone to attribute a causal character to one of the phenomena without requiring valid, scientifically grounded evidence. This remark applies, for example, to the focal theory, which attributes certain peripheral diseases to morbid changes in the tonsils and the teeth; to Wittmaack's "theory of pneu-

1. This historical review has been compiled from that of A. Denker and O. Kahler (Handbuch der Hals-Nasen-Ohrenheilkunde mit Einschluss der Grenzgebiete, Berlin, Julius Springer, 1926).

matization," whereby the development of air cell systems of different size is ascribed to infantile otitis, and to the supposition that dental caries can be traced to the action of certain carbohydrates, notably "candy." Examples might be multiplied.

When a particular view (of hypothetic nature) regarding cause and effect has once been established in the literature, particularly in textbooks, it is liable, owing to faith in authorities, to get firmly rooted in consciousness. In the course of one's work one may discover that certain phenomena and symptoms observed clinically do not accord at all with the prevalent beliefs; that others cannot be explained in the light of the accepted theory, and that others, again, do not need it for their explanation. For all that, it goes against the grain to return to the starting point and to reconsider whether one is really concerned with a case of cause and effect and, if so, whether it should not be viewed in the reverse direction, what is now regarded as cause being actually an effect, and vice versa.

I do not claim that the cause and effect relation between chronic otitis and cholesteatoma has been actually proved to be just the reverse of the present view. I am not in a position to show that cholesteatoma is the primary factor and chronic otitis the secondary. Here I propose merely to touch on certain phenomena relating to the occurrence of cholesteatoma, on the one hand, and to the symptom complex of chronic otitis, on the other, in order to indicate the superficial way in which these problems have hitherto been investigated. The flimsy foundation for the present belief that cholesteatoma originates from chronic otitis will thus be made abundantly clear.

Viewed logically, the emphatic statement of Gruber and Troeltsch that cholesteatoma of the ear is not a real tumor topples to the ground. Obviously, the fact that cholesteatoma of the ear is of far more frequent occurrence than cholesteatoma of the pia mater does not per se rule out the possibility that both may be genuine tumors. Nor is this view excluded by the indication that the development of cholesteatoma might be due to the presence of an external irritant. The mere mention of the carcinoma of the lips of pipe-smokers will at once make the matter clear. Thus, even if chronic otitis were proved to be the precipitating factor in the development of the cholesteatoma, the tumorous character of the latter could not be considered to have been thereby ruled out. Moreover, as will be presently shown, it has not even been demonstrated that chronic otitis is chronologically the primary phenomenon and cholesteatoma the secondary. This, of course, must be postulated in vindicating the theory that the cholesteatoma is caused by chronic otitis.

On the other hand, even if valid grounds could be adduced for denying the tumorous character of cholesteatoma, this obviously

would nowise imply that chronic otitis is the cause or even the primary phenomenon. In short, even if cholesteatoma is not a genuine tumor, it may nevertheless be chronologically the primary formation. In that case chronic otitis cannot possibly be the cause of cholesteatoma.

In any case, there is some difficulty in explaining the origin of a growth, whether tumorous or nontumorous, proceeding from flat epithelium at a place where such epithelium is not "normally" found. It was long supposed that flat epithelium never occurred in the mucosa of the tympanic cavity unassociated with chronic otitis and that cholesteatoma was never found in the aural region except in that connection. It could therefore be considered *naheliegend* to accept Bezold's unproved view that flat epithelium migrates from the auditory meatus through the marginal perforation of the tympanic membrane into the middle ear. Habermann's interpretation of histologic preparations showing a direct communication between the cholesteatoma matrix and the flat epithelium of the auditory meatus could, for the same reason, be pleaded in support of the theory that such epithelium migrates through the marginal perforation.

However, the startling discovery that islands of flat epithelium (besides fully developed cholesteatoma) might appear in the mucosa of the middle ear without the simultaneous presence of marginal perforation or chronic otitis should have aroused suspicion regarding the validity of Bezold's hypothesis and Habermann's "proofs"—thus forcing one to the conclusion that a neoplastic etiologic factor must be reckoned with as a possibility.

The adherence to Bezold's hypothesis as the sole alternative must be ascribed to inertia and to innate reluctance to abandon "knowledge" acquired from standard books. On the other hand, evidence that this hypothesis was wrong could not be produced. Thus, even his patently incorrect statement that when chronic otitis and marginal perforation of the tympanic membrane are associated with cholesteatoma the chronic otitis always manifests itself first was left unrefuted.

When, in view of the absence of tympanic perforation, it is inconceivable that flat epithelium could have migrated from the auditory meatus to the middle ear, one must try to account for the presence of flat epithelium, which in certain cases has actually been observed as islands interspersed in the mucosa or in the form of a fully developed cholesteatoma.

It seems strange that in cases of this description, too, for reasons based on analogy, the possibility of a neoplastic origin of the existing cholesteatoma of the ear is still being denied. Such evidence might have been used with equal cogency to declare that in all cases choles-

teatoma of the ear is a tumor as soon as it was shown that a genuine cholesteatomatous tumor might appear in the ear as well as in the pia mater.

A number of hypotheses are being set up to explain how flat epithelium may occur in the middle ear in cases in which the tympanic membrane is normal. Thus the origin of cholesteatoma has been attributed to such different factors as epithelial scission from a temporarily intruding funnel-shaped Shrapnell's membrane (Wittmaack), *Keimversprengung* (germ dispersal—Körner, Manasse and Link), metaplasia (Wendt) and trauma sustained at birth or later in life (Kelemen).

Whichever of these hypotheses is accepted, or if actually all of them should be found correct, it is obvious that neither marginal perforation of the tympanic membrane nor chronic otitis is a *sine qua non* for the origin of cholesteatoma. It is also clear that a neoplastic etiologic factor cannot be ruled out—nor can it be proved to be present—in a single case in which cholesteatoma has been observed.

This statement is nowise affected by the indication that metaplasia or growth of the germ—whatever its origin—of a larger or smaller observable cholesteatoma may have been caused by chronic otitis.

In the light of the aforementioned facts it seems justifiable to renew the discussion of the question as to what is cause and what is effect in the relation between cholesteatoma and chronic otitis with marginal perforation of the tympanic membrane. Clinically, at any rate, this is incontestably a common combination.

As regards the question whether there is a connection between marginal perforation and chronic otitis, it should be noted that, unlike central perforation of the tympanic membrane, marginal perforation, as the textbooks indicate, has never yet been observed by anybody to occur in close association with acute otitis. Logically, therefore, it must be inferred that acute otitis is not responsible for marginal perforation of the tympanic membrane, as otherwise the connection could scarcely have eluded observation.

How does marginal perforation of the tympanic membrane then arise? Theoretically, it might be caused by some infection which has a chronic tendency from the outset. But it may also be due to some process of a noninfectious nature. It is conceivable that the perforation may be caused by the destructive effect of an expansive growth, similar to the formation of a fistula of the labyrinth or the breaking-down of the internal cortex, with exposure of the meninges, which occurs under pressure of a cholesteatoma.

It is nowise my intention to contend that cholesteatoma, by its destructive action, demonstrably gives rise to the marginal perforation

of the tympanum, but I desire strongly to emphasize that such a process would be analogous to other bone destructions caused by expansive growth..[2]

From this point of view one can, at any rate, more easily account for a symptom-free development of such a serious formation as a fistula of bone of the innermost part of the external auditory meatus, where infections otherwise cause intense periosteal pain. It also explains why, according to the textbooks, no one has seen a marginal perforation of the tympanic membrane develop in immediate connection with acute otitis. In fact, one is under the illusion that its development cannot be seen at all. One is so wrapt up in one's idea that marginal perforation of the tympanic membrane is a sure indication of chronic otitis that the mere observation of such a perforation at once arouses the suspicion that it had been preceded by otitis with a chronic course. In the face of the patient, who in many cases is quite unaware of any disease in the ear, one asserts that one knows better, that he must "for a long time" have suffered from an aural disease. Against the patient's emphatic statement that he has never observed any secretion in the external auditory meatus, one maintains that such a secretion must have "previously" occurred. One goes to such lengths in self suggestion that when a marginal perforation is detected in a patient whom, after a recent examination, one has registered as having a normal tympanum, one actually disavows the correctness of one's own observations. In short, one proceeds from the assumption that the previous otoscopic examination must have been defective, since it failed to "detect" the chronic otitis which, one imagines, must have already existed at the time. No wonder that the textbooks assure their readers that no one has ever seen a marginal perforation develop.

If, instead, one sets out from the possibility that the marginal perforation of the tympanum may have been produced from within by the destructive action of a growing cholesteatoma unassociated with otitis (which when it occurs in connection with a marginal perforation may be an intercurrent infection), one will often see marginal perforations develop. And one will frequently see such perforations heal when the desquamated flat epithelium content has been spontaneously discharged into the auditory meatus. In certain cases one will see this "performance" several times repeated (as I myself have), until it finally issues in a permanent marginal perforation of the tympanum, tending to increase in size. It may be added that the tympanic membrane at first need not be involved at all in the fistula,

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2. Diamant, M.: Acute or Chronic Otitis, Acta oto-laryng. **25**:507, 1937.

but will take part in its development after the perforation has been enlarged.

If this view is accepted, it will be understood that the marginal perforation of the tympanum is neither "chronic" nor "otitis." It will enable one, however, to throw light on a number of otherwise inexplicable phenomena. One can then explain the detection of a cholesteatoma behind a clinically normal tympanic membrane. One can explain the less frequent observation of cholesteatoma in cases of central perforation by pointing out that a not yet manifest cholesteatoma might be associated with acute otitis which is causing central perforation. One will not need to resort to the artificial explanation that cholesteatoma cannot penetrate from the external auditory meatus through a central perforation, but only through a marginal one; whilst the so-called traumatic cholesteatoma can be accounted for by such relatively slight operative procedures as paracentesis. One can explain Jorgen Moeller's[3] case of "genuine" cholesteatoma complicated by acute otitis. Finally, one can explain many of the cases in which "traumatic" cholesteatoma is reported to have developed after mastoidectomy or, as just indicated, merely after paracentesis. The germ of cholesteatoma may, in fact, have already existed at the time of the operation for mastoiditis of independent origin (Diamant[2]). The cholesteatoma may not begin to grow until a later stage, occurring immediately or some time after the healing, thus obliging the patient again to consult a doctor. Now that the cholesteatoma can more easily make its way toward the operative opening, its destructive effect may possibly be avoided. There is nothing to gainsay the supposition that the carrier of a cholesteatoma germ may be more liable than others to contract acute otitis (see later comment concerning this point). Nor, of course, is there implied a denial of the possibility that cholesteatoma may develop traumatically in the aural region.

Acceptance of the view that cholesteatoma is the primary factor in the development of the marginal perforation and that "chronic otitis" supervenes does not necessarily involve assenting to the belief that cholesteatoma is a genuine tumor. The question as to the cholesteatoma germ that originates and develops at a place where flat epithelium is not usually found still remains open and unsettled. The foregoing discussion has shown that the question whether the marginal perforation is related to the cholesteatoma need nowise be connected with the view that it is a genuine tumor. It has also shown that a denial of the theory that it is a genuine tumor does not automatically imply that cholesteatoma is caused by chronic otitis with marginal perforation of the tympanic membrane.

3. Moeller, J.: A Case of Primary Cholesteatoma Complicated by an Acute Otitis, Acta oto-laryng. **1**:309, 1918-1919

As already indicated, I do not claim that without further investigation the hypothesis set forth and the observations reported here can be substantiated. Scientifically convincing evidence can scarcely be based on personal observations which cannot, somehow or other, be reproduced. Though some advance has been made in the pictorial reproduction of the tympanum with the camera's eye, the latter does not enable one to reproduce any regions in sufficient detail to show the successive stages of the development of the marginal perforation. This, however, should not prevent one from realizing that the view according to which cholesteatoma develops in consequence of chronic otitis with marginal perforation is quite as unproved as the theory now advanced that the marginal perforation as well as the supervening infection which is termed chronic otitis may be primarily caused by cholesteatoma.

On the ground of the latter hypothesis, I have for about ten years refrained from making the diagnosis "chronic otitis." I have, instead, diagnosed "perforatio membrani tympani centralis" (or "marginalis"), adding, as the case might be, "cum suppuratione," or "cum polype," or "cum cholesteatomate." This seems to me to be a more correct designation of a disease the classification of which is actually based on the appearance of the tympanum, the secretion and the occurrence of polypi or of cholesteatoma.

To this, it may be desirable to add a few remarks on a matter which is not merely of nomenclatural interest. One still often finds in the literature the terms "normally pneumatized os mastoideum" and "retarded cellular system." Such expressions, too, seem to me to indicate an unduly uncritical and undifferentiated attitude toward previously acquired book knowledge. I consider myself to have satisfactorily ascertained and shown[4] that the air cell system of os mastoideum varies in normal material from no cells up to systems exceeding in size any which one finds in persons suffering from otitis. The distribution of the different size groups according to frequency shows the typical picture of a normal curve.

If, for a moment, the discussion is transferred to an analogous subject, one finds that human stature, in a normal material, varies about an average figure with a frequency distribution likewise represented by a normal curve. Evidently, however, it is a misnomer to speak of persons showing the average height as being of normal stature. It is a similar misnomer to speak of a mastoid process with an air cell system of average size as "normally pneumatized." In re-

4 Diamant, M.: Otitis and Pneumatisation of the Mastoid Bone: Clinical-Statistical Analysis, Acta oto-laryng., 1940, supp. 41; Otitis and Air Cell Systems, Arch. Otolaryng. **34**:24 (July) 1941.

gard to a person who is shorter than the average but whose height falls within the range of the normal curve within $\pm 3\ \sigma$, it is incorrect to say that he is stunted. It is equally incorrect to speak of a "retarded" air cell system in regard to a cellular system of small size—for such small systems may be found in normal persons right down to no cells. The analogy differs only in the fact that in Sweden the statures of mankind, according to the normal curve within $\pm 3\ \sigma$, range between 163.2 and 193.2 cm. at the age of about 20, according to an article by Broman, Dahlberg and Lichtenstein in the pocket diary published by the pharmaceutical factory Astra; whereas, according to my own investigation, the size of the cellular system varies from 0 to about 30 square centimeters (on a roentgenogram in lateral projection).[5]

These observations on the size of the air cell system are intended to serve as an illustration of the fact that cholesteatoma is usually found in an ear with a small cellular system. I showed (in 1940) the distinct connection between different sizes of the cellular system and different forms of otitis. From this investigation it is evident that marginal perforation of the tympanum occurs precisely in connection with cellular systems which, on an average, show the smallest size. As this is in fact the case, it seems reasonable to suppose that development of cholesteatoma when it occurs in the middle ear is somehow connected with the factors which entail the development of the smallest cellular systems. (This obviously applies even if one accepts the theory that marginal perforation of the tympanum is caused by the destructive action of a cholesteatoma.) It has been shown by Dahlberg and Diamant[6] that heredity plays the principal part in the development of the size of the air cell system. One might therefore presume that it likewise plays some part in the origin of the cholesteatoma germ and the development of cholesteatoma. This, however, is not per se a given fact. The implication is that the cholesteatoma germ and developed cholesteatoma should actually have been observed to occur more frequently in cases in which there is marginal perforation of the tympanum and in which the cellular system is small than in cases in which the cellular system is large and in which there is no marginal perforation. The real truth might be that the cholesteatoma which results in a marginal perforation is placed on record because the accompanying symptoms compel the diseased person to consult a doctor.

Thus, it will be necessary to examine normal material with special regard to the occurrence of the cholesteatoma germ and the developed

5. For further particulars concerning this subject, see Diamant, M.: Risks of Chronic and Acute Otitis, Acta oto-laryng., to be published.

6. Dahlberg, G., and Diamant, M.: Hereditary Character of the Cellular System in the Mastoid Process, Acta oto-laryng. **33**:378, 1945.

cholesteatoma. The difficulties, however, are great in that the cholesteatoma germ, theoretically, may be represented by a single flat epithelium cell (*Keimversprengung*) and may therefore elude detection even in a thorough histologic examination of sections of the tympanic region and its environment.

Moreover, the growth of a cholesteatoma germ in the tympanic cavity and its adnexa may possibly be due to causes of an entirely local nature, without any hereditary basis. The marginal perforation of the tympanum may thus be traced (1) to hereditary factors giving rise to the cholesteatoma germ in connection with the development of the small cellular system and (2) to an environmental factor stimulating the growth of the cholesteatoma. It should be emphasized that the environmental factor need not necessarily be of otitic character.

In conclusion it must be reiterated that I nowise claim to have presented cogent evidence in favor of the statements made here regarding the etiologic factors of cholesteatoma or the relation of this growth to marginal perforation of the tympanic membrane and chronic otitis. For the sake of completeness, it should be pointed out that the conditions in this respect need not always be uniform. It is theoretically conceivable that, whereas in certain cases the cholesteatoma is the primary phenomenon and the marginal perforation secondary, in other cases quite different conditions may exist.

The object of this discussion has been to appeal to those of my colleagues who grapple with these difficult problems to try to emancipate themselves from, and to work untrammeled by, the hitherto accepted, but as yet unproved, hypotheses according to which it is supposed that chronic otitis with marginal perforation of the tympanic membrane is primary to and the cause of cholesteatoma of the ear, which is asserted to be nontumorous.

TEST FINDINGS BEFORE AND AFTER FENESTRATION OF THE LABYRINTH

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WE NEED NOT dwell at length on the importance of carefully selecting the otosclerotic patient for the fenestration operation. Certainly the conscientious operator does not care to handle the poor risk, and likewise the otologist referring the patient does not want to recommend the operation only to be reversed by the operator. Their greatest protection and the best interests of the patient demand a particularly careful preliminary study of the hearing. Not one or two tests are adequate, but, as in all cases of deafness, a considerable amount of evidence must be accumulated and evaluated. The tests discussed in this article are all considered necessary and important.

The following items, of importance in selecting the otosclerotic patient for operation, will not be discussed, as they are not considered pertinent to the title of this presentation: history of the patient, age, recuperative powers, rate of progression of hearing impairment, psychologic aspects and occupation.

AUDIOGRAMS BEFORE AND AFTER OPERATION

The audiometer curve of ostosclerosis varies somewhat, according to the advancement of the pathologic process. In cases in which the loss of hearing is measured early, there is typically greater loss for the lowest frequencies and less loss for the middle and highest frequencies. In cases in which the otosclerosis is moderately advanced, the audiometric curve is nearer a straight line, with still a slightly greater loss for the low frequencies and a probable dip for the 4096 frequency. In cases in which otosclerosis is well advanced, there remains the considerable loss for the low and middle frequencies while the loss for the highest frequencies (4096 and 8192) is still greater, the curve taking a sharp downward trend at these high levels.

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In table 1 are shown the audiograms of the affected ears of 10 consecutive patients with typical otosclerosis—as proved by the finding of a fixed stapes at the operation.

An analysis of these audiograms reveals that in general the losses of hearing for the 128 and 256 frequencies are almost exactly the same, the losses for the 512 and 1024 frequencies are almost exactly the

TABLE 1.—*Results of Audiometric Tests Made in 10 Successive Cases of Otosclerosis*

Frequency	LOSS OF HEARING IN GIVEN CASE, IN DECIBELS										Average Loss
	147	148	149	150	151	152	153	154	155	156	
128	52	54	47	50	33	43	50	45	48	45	46.7
256	50	55	50	47	37	43	50	45	43	45	46.5
512	57	58	50	58	43	50	47	45	48	45	50.1
1024	57	70	50	58	38	53	48	45	45	45	50.9
2048	55	63	30	53	35	50	50	35	33	53	45.7
4096	50	53	35	47	33	35	60	55	48	58	47.4
8192	40	37	25	60	13	55	65	35	35	70	43.5

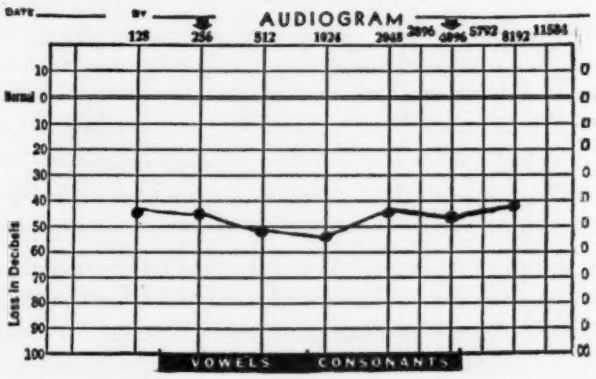

Fig. 1.—Average audiogram based on 10 typical cases of otosclerosis in which this disease was proved to be present by the finding of a fixed stapes at operation in each case.

same but slightly greater than those for the two lower frequencies, the loss for 2048 is slightly less than the losses for lower frequencies, the loss for 4096 is somewhat greater again, while that for 8192 is the lowest of the losses recorded for all frequencies.

Although the 10 cases were consecutive, they might be considered somewhat unusual in that in none of them was there the considerable loss of hearing for the two highest frequencies that is so commonly met with in the cases of otosclerosis. Frequently there is a loss of 75 or 80 decibels for the 4096 frequency and a loss of 80 to 100 decibels for

the 8192 frequency without a proportionate loss for the middle frequencies (1024 and 2048). This great loss restricted to the highest frequencies means deterioration of the auditory nerve at those levels, and it may mean that this deterioration has extended, in a lesser degree, to the 2048 frequency or lower. On the other hand, total or nearly total loss for the two highest frequencies may be found in cases in which the loss for 2048 is apparently entirely of a conductive type, as indicated by little or no loss by bone conduction and no great loss by air conduction, and by a considerable improvement in hearing for this frequency after the fenestration operation.

An analysis of the air conduction curves in such cases of otosclerosis is helpful in attempting to determine how much of the hearing loss is conductive and how much perceptive in type. It cannot be conclusive, however, particularly as one does not know how great a loss of hearing of the purely conductive type a person may have. When the

TABLE 2.—*Decibel Improvement Obtained by Operation in 6 Typically Successful Cases*

Frequency	IMPROVEMENT OF HEARING IN GIVEN CASE, IN DECIBELS						Average Improvement
	147	148	150	152	155	156	
128	38	25	20	23	35	25	27.7
256	35	30	23	33	30	27	29.7
512	43	30	28	35	25	25	31.
1024	38	30	23	32	40	25	31.3
2048	40	28	32	30	33	27	31.7
4096	25	15	0	0	30	0	11.7
8192	0	0	0	0	0	0	0

hearing of an otosclerotic patient is improved by operation from the 55 decibel level to the 20 decibel level in the low and middle frequencies, we assume that the preoperative loss of hearing for those frequencies was of the conductive type, and when the operation in this patient produces no improvement in the 4096 and 8192 frequencies, it suggests that the preoperative loss of hearing for those frequencies was perceptive in type.

AUDIOMETRIC CURVES IMPROVED BY OPERATION

The frequencies for which hearing is improved by the fenestration operation are those in the low and the middle range.

Fortunately, the greatest amount of improvement is in the hearing for those frequencies that are in the conversational range—512, 1024 and 2048.

Table 2 shows the typical improvement and the amount with respect to the various frequencies in several selected successful cases.

The figures show that the hearing for the three speech frequencies was improved to approximately the same extent, being very slightly better for the 2048 frequency than for any of the others. The improvement for the lowest frequencies is nearly as great as that for the middle ones, with slightly less improvement at 128. The improvement for 4096 is slight and, as is shown in the table, is nothing in half of the cases. In these cases there was no change of hearing for the 8192 frequency in spite of the fact that the preoperative loss of hearing for that frequency was on the average less than that for any of the other frequencies. The figures in this table are shown only to indicate the typical improvement that is noted at each of the test frequencies in highly successful cases. They by no means show the average improvement obtained in a large series of cases.

BONE CONDUCTION HEARING

One recognizes, above all things, that good bone conduction is a prerequisite for operation. Yet, one is faced often with inadequate and indeterminate testings to reveal the status of hearing by bone conduction. Masking is essential, and it should be measured masking; that is, masking raised in decibel steps to just the required amount but never to a point where spreading to the opposite or tested ear is observed. Thus one can avoid flooding the head with sound, which puts into the discard all measured testing. Important also is the use of a masking tone that is just below the tone used in the tested ear. We believe that ultimately the present mixed noise used for crowding out the untested ear will be discarded. Fortunate is the examiner who has two audiometers, one of which can be used as the masker. The octave below is adequate if one has not a sweep oscillator, which will bring the masking tone ideally nearer, but still below, the tone used in testing.

Masking certainly should be done in the untested ear if the hearing of this ear is 30 to 40 decibels better than that of the tested ear. This applies to both bone conduction and air conduction testing. One can conveniently use either a bone conduction or an air conduction receiver in order to mask, depending on whether one is testing the opposite ear with air conduction or with bone conduction. A most exact method is to use a bone conduction masker when testing with air conduction and vice versa. Then the testing air-conducted or bone-conducted tone may be applied to the untested ear and the loudness of the masking sound can surely be raised to a level to dampen it out. Thus one can be sure of using only just the needed amount of masking, avoiding flooding the head with sound.

"Hearing across the head" can be an actuality and must be constantly kept in mind and guarded against. One cannot altogether de-

pend on the patient's reporting as to which ear is doing the hearing. Herein lies the value of using different pitches for masking and testing. The patient can identify each.

One consideration in bone conduction testing, whether with audiometer or with fork, is the avoidance of air-borne sound. The most definite assurance in this matter is to run off a reporting by the patient of loudness of levels at which different pitches are heard when the bone conduction receiver or the fork is held in the bone conduction position but not in contact with the bone. It is well to make a record of the findings so as to compare them with those obtained later with the routine bone conduction technic. If at any pitch the two reportings coincide, the examiner cannot tell what the patient reported—air conduction or bone conduction.

The matter of bone conduction hearing before and after operation is interesting. Since usually only the patients with good bone conduc-

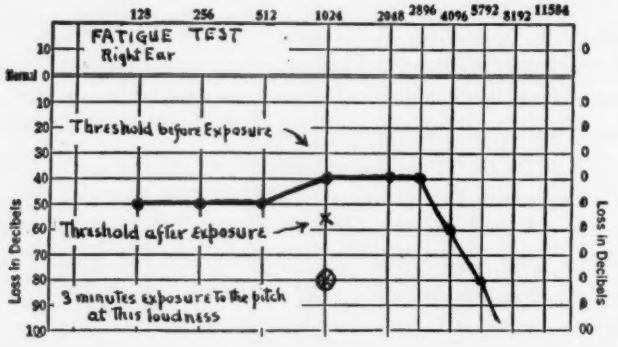

Fig. 2.—Recording of the fatigue test. The right ear and a pitch of 1024 cycles per second were used. The threshold at the start was 40 decibles. The ear was exposed to the frequency mentioned for three minutes at 80 decibels. After this three minutes the threshold was 55 decibels; after four minutes it was 50 decibels; after five minutes it was 45 decibels; after six minutes it was 40 decibels. Conclusion: There had been 15 decibels fatigue, with recovery in three minutes.

tion hearing are operated on, there should be little or no change in this hearing. However, errors of testing may be made, for the patient whose air conduction is benefited by operation has better hearing of air-borne sound when one is testing for bone conduction hearing. Again, one must take a frequency run with the bone conduction receiver "just off" the mastoid, to see what may erroneously be reported for bone conduction hearing.

THE FATIGUE TEST

Auditory fatigue does not show up in catarrhal deafness nor in early otosclerosis but it does appear in all types of nerve deafness. The test needs usually to be performed with but one or two frequencies, preferably in the speech-hearing zone. It is well to pick a pitch

for which there is sufficient hearing to allow for delivering of a loudness 40 decibels above the patient's hearing threshold. This loudness is left on for three minutes, and then the patient's threshold is retested every thirty seconds. Recovery does take place, either slowly or rapidly. The test gives another means of separating nerve deafness from conductive deafness.

THE GELLÉ TEST

The Gellé test is believed to be not as popular in this work as it should be, and yet one should depend on it greatly. Certainly, a normal result of this test suggests that if an otosclerotic process is present it has not attacked the stapes. We definitely believe, from both testing results and from what is seen at operation, that there can be varying

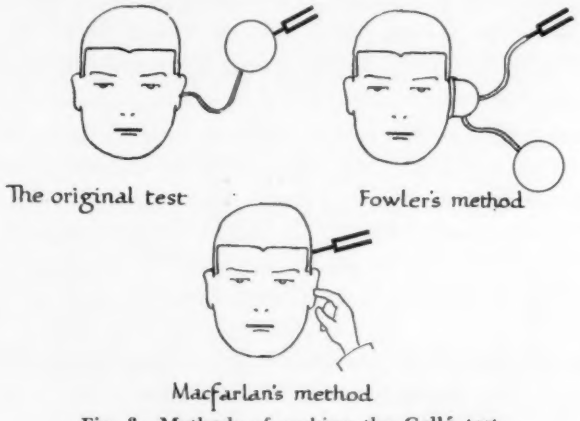

Fig. 3.—Methods of making the Gellé test.

degrees of fixation. In testing we have found the Gellé test eliciting a more and more positive response as the disease progressed. When some fixation is surmised and when the rest of the picture points to otosclerosis, one is more than ever influenced toward early operation. Late, complete fixation accompanied by poor hearing of bone conducted sound means that the case has gone too far.

We submit a convenient modification of the classic Gellé technic. The old technic is often limited by reason of the patient not being able to hear the sounding fork placed against the bag. Further than this, the air escaping out of the auditory canal when pressure is applied interferes with hearing. In the attempt to avoid this leakage, too much pressure of the tip being pushed into the ear produces a premature Gellé effect before reporting begins. The new technic is to hold the butt of the fork on the mastoid process and to use the index finger to exert pressure on the column of air between the meatus and the drum.

Intensity matching, loudness balance, recruitment (call it what one may) gives a definite demarcation between "operable and "inoperable" loss of hearing in patients with otosclerosis. The test is simply an attempt to reach equal loudness of sound in each ear by raising the intensity. It cannot be accomplished if the deafness is due to an obstructing process but can be if there is nerve deafness. Fortunately, there is usually enough disparity between the two ears at some pitch to make the test possible. Fowler's very original discovery will tell one when the end organ is being choked by otosclerotic disease.

Otosclerosis in the beginning and in the "operable" stage is clinically a conductive deafness and loudness balance is impossible. Fowler believes this to be the case always in otosclerosis, yet we seem to find it otherwise when the disease has advanced to the point where the perceptive apparatus is damaged. Of course, in this stage the hearing by bone conduction is also down and there will be no benefit from operation. Degrees of intensity matching are seen, just as degrees of stapes fixation are seen.

The foregoing statements on loudness balance are compromised by the fact that the high frequencies may be choked in the cochlea if otosclerosis is in the advanced stage. Then there could be loudness balance in these frequencies typical of perceptive deafness, while the lower pitches, unaffected as yet by the cochlear choking, would still give the finding of absence of matching as seen in conductive deafness.

HEARING OF SPEECH BEFORE AND AFTER OPERATION AND ITS CORRELATION WITH AUDIOMETRIC HEARING

The desirability of obtaining a record of the hearing of speech as well as of the hearing of the pure tones of the audiometer before and after operation in all cases of otosclerosis is being recognized more and more, and speech and pure tone audiometers are being developed to correlate accurately such measurements of hearing.

Now one has a definite and metered way of testing the hearing of speech; and the thresholds, as well as the efficiency of hearing at different loudness levels, can be sharply determined by interpretation tests with a phonograph audiometer.

An unamplified phonograph may be brought at small cost. The turntable should be electrically driven, the pickup should be the crystal type and there should be an intensity trimmer on the instrument. The output of the phonograph is plugged into the microphone outlet of an audiometer, and, thus used, the audiometer supplies the necessary amplification and a decibel meter. The patient hears the speech coming from an ear phone. The record should be one in which the speech is recorded at a definite constant intensity or loudness. The

decibel meter allows one to change the loudness in decibel steps, so that exact threshold readings for speech hearing are obtained. The record developed by one of us contains on one side a list of numbers and on the reverse side a series of common monosyllabic words. Preferably, the number side is used for threshold testing, while the common word list is used in testing the efficiency with which speech is

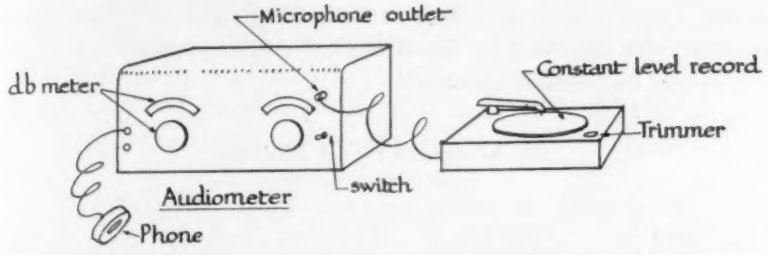

Fig. 4.—Simple and practical phonograph-audiometer for determining the hearing of speech. Phonographic speech is fed into the microphone circuit of the audiometer and is varied at will as to loudness by the decibel meter dial.

Dr. Macfarlan's Test Method

Record No. 1

55--Two Digit Numbers--Constant Level

48	85	62	41
23	66	32	81
66	64	38	68
41	58	43	21
81	38	52	
68	18	51	
21	23	22	
15	48	56	
43	63	65	
31	83	25	
24	32	16	
56	85	84	
55	63	32	
54	18	64	
88	24	48	
42	88	23	
23	25	66	

Fig. 5.—Numbers recorded on a phonograph record at constant intensity.

interpreted at various levels of intensity. It is surprising to find that the patient's reporting is as accurate for speech as it is for pure tone. (A most careful recording of a flat intensity record is essential.)

Walsh and Silverman[1] have made a most valuable and original contribution to speech audiometry as carried on at levels above thres-

1. Walsh, T. E., and Silverman, S. R.: Diagnosis and Evaluation of Fenestration, Laryngoscope **56**:536 (Sept.) 1946.

hold. Their work analyzed the improvement in interpretation as speech sounds were made louder and louder. The patients with conductive deafness showed continuously better hearing as loudness was increased, whereas with nerve deafness a level of loudness was reached at which there was an end of the patient's maximum efficiency. Beyond this, greater loudness did not make for better hearing. The test is invaluable for differential diagnosis, particularly in cases of otosclerosis. Here is another means of separating cases in which the patient would be benefited by operation from those in which the disease has advanced to cochlear damage.

Dr. Macfarlan's Test Method

Record No. 2

48 Common Monosyllables--Constant Level

Boy	Green	Light	Play
Good	Bird	Hard	Milk
Cold	Blue	Burn	Stop
Hand	Lip	Horse	Train
Go	Ear	Look	Walk
Eat	Long	Girl	Man
Give	God	Head	Love
Love	By	Bear	Sun
Door	Big	Low	Tree
Child	Box	Black	Two
Drink	Lost	Ball	Now
Egg	Book	Home	You

14 Monosyllables for Pre-school Child Test

Constant Level

Box	Dog	Girl	Boy
Apple	Bird	Door	Ball
Ball	Boat	Book	
Moon	Boy	Bell	

Fig. 6.—Monosyllabic works recorded an a phonograph record as constant intensity.

One cannot but compliment Lempert and his followers on the high goal which they set for themselves in choosing the objective of social and economic restoration of the hearing of speech. Is the goal too high, and is it excluding cases in which there is a possibility of obtaining an improvement of hearing in persons who suffer from more severe loss? Surely the deafened will welcome any hearing improvement. Even though the benefit may not be so great as to permit them to discard a hearing aid, is not much to be gained by the better hearing which the aid will produce after the operation? In considering these questions, two things must be understood—(1) the loudness of normal speech and (2) the point where speech-hearing disability begins. In average conversational situations the speech of a person

with normal hearing is of a loudness of 40 decibels above audible sound. With the hard of hearing, difficulties in the hearing of speech are definitely noticed at a 20 decibel loss in interpretation testing. The person with a 20 to 25 decibel loss will say, "I can get along under most circumstances. It is only once in a while that I encounter a low speaking voice, or one in which articulation is slurred or poor. Then, too, I am bothered with persons speaking too rapidly; I do not have time to catch all that they say." Cross talk difficulties appear with this loss, but telephone conversation is invariably satisfactory. At a 30 decibel loss, lapses of auditory attention begin to appear. It is too much of an effort to keep trying to hear. At this stage these people begin to withdraw from, and avoid, as many speech contacts as pos-

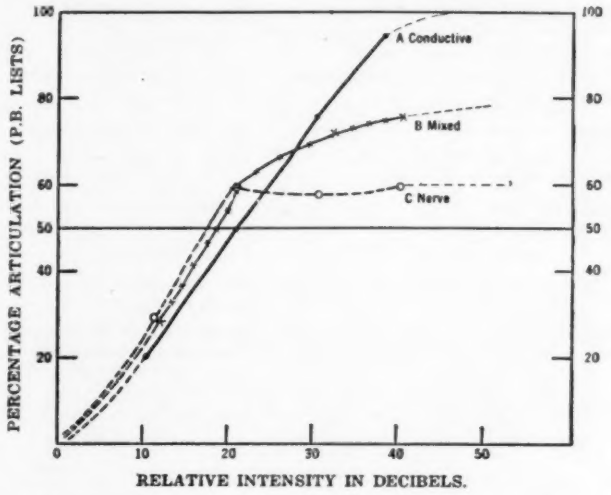

Fig. 7.—Articulation for conductive, mixed and nerve type deafness (from Walsh and Silverman.1)

sible. Fear of making mistakes in understanding begins. Office workers are afraid they will not get correctly the directions or the dictation given them. Sensitiveness and nerve strain appear. It is at this point that a hearing aid and lip reading jump the patients back over the barriers of lapses of attention or mistakes of hearing. At a 20 decibel loss or better, to which the hearing aid boosts them, they no longer have to make the effort to hear. The critical zone, then, can be said to be in the neighborhood of the 30 decibel loss. It seems to be a matter of simple mathematics to say that if an otosclerotic patient with a 50 decibel loss of hearing is helped to the 30 decibel loss by an aid, the fenestration operation, even if it reduced the loss but 10 decibels, would be a great boon.

It is a source of great satisfaction to see the workers in fenestration lead the way to an appreciation of the importance of speech hearing. In considering both rehabilitation and the patient's point of view, it had to be so. Frequency audiometry, with its deduction as to the capacity to understand speech, has long been misleading as to the hearing of speech. It seems advisable to accept the dictum that one cannot expect to know what the hearing of speech is from the study of an audiogram.

In tables 3 and 4 are recorded the speech reading and the audiometric reading before and after operation of each of 10 consecutive otosclerotic patients who were subjected to the fenestration operation.

TABLE 3.—*Speech Readings Before and After Operation*

	LOSS OF HEARING FOR SPEECH IN GIVEN CASE, IN DECIBELS									
	147	148	149	150	151	152	153	154	155	156
Before operation	60	70	50	65	50	50	65	60	55	60
After operation	30	40	35	35	30	30	45	25	25	35
Net improvement	30	30	15	30	20	20	20	35	30	25

TABLE 4.—*Audiometric Readings Before and After Operation (Average for the 512, 1024 and 2048 Frequencies)*

	LOSS OF HEARING FOR SPEECH IN GIVEN CASE, IN DECIBELS									
	147	148	149	150	151	152	153	154	155	156
Before operation	57	64.6	43.3	56.3	38.3	51	48.3	41.6	45.3	47.6
After operation	15	35	36.6	28.3	28.3	18.3	30	16.6	11.6	21.6
Net improvement	42	29.6	6.7	28	10	32.7	18.3	25	33.7	26

A study of these postoperative findings reveals considerable variation in the comparative improvements to pure tones and speech, so that accurate correlating of one with the other is difficult or impossible. One cannot predict from the frequency graphs taken after operation what change has taken place in the hearing of speech. The graph may be better as compared with the speech reading, or the opposite may be found. In general it will be discovered that the speech level of hearing after operation as recorded in decibels is not as good as the pure tone level. In these 10 cases the average speech level after operation was 33 decibels, while the average pure tone level was 24.

Further comparisons may be made in the speech and audiometric readings in connection with the various frequencies involved. Has it

been found that the hearing of speech after operation has any relation to the particular audiometric frequency that indicates improved hearing? In table 5 is shown, in decibels, the amount of improvement found in speech and in pure tone hearing after operation in the same 10 cases; also, the improvements obtained for the three speech frequencies individually.

It has been mentioned by observers of patients who had been operated on that a postoperative improvement of hearing for only the one frequency 2048 will give a substantial improvement of the practical hearing of speech, and this appears to be substantiated by analysis of these few cases. However, an improvement obtained for the 1024 frequency will give a further rise in the hearing of speech

TABLE 5.—*Decibel Improvement Found After Operation*

	IMPROVEMENT IN GIVEN CASE, IN DECIBELS									
	147	148	149	150	151	152	153	154	155	156
Audiometric improvement	42	29.6	6.7	28	10	32.7	18.3	25	33.7	26
Speech improvement	30	30	15	30	20	20	20	35	30	25
Improvement at 512	43	30	0	23	8	35	15	25	25	25
Improvement at 1024	38	30	10	25	13	33	5	25	42	25
Improvement at 2048	45	28	10	30	10	30	35	25	33	28

and an improvement for the 512 frequency, in addition to the improvements for 2048 and 1024, will give a still further boost to the hearing of speech.

TINNITUS

One must not omit consideration of tinnitus when it is present in any deaf patient, for tinnitus alone may greatly influence hearing in the zone where it lies. A note on the audiogram should show the region of the predominate pitch present in the noise. Quantitative measurement of the tinnitus is possible only by testing the hearing both when the noise is absent and when it is present. Subtraction of the two findings gives an acceptable index.

VESTIBULAR TESTS

Since one feels obliged to collect all available evidence on the otosclerotic patient, the vitality of the vestibular apparatus must be studied. It either has or has not been influenced by the disease. Its reactions will surely give a clue to the extent, if any, of the involve-

ment, and this may be an added item in deciding for or against operation. Otologists in general have different preferences for vestibular tests, but whether the tests be turnings or fractional or massive douchings, the information sought for is the same: Is, or is not, the vestibular apparatus involved?

VERTIGO AND THE FISTULA TEST AFTER OPERATION

All performing fenestration have had considerable experience with vertigo and fistula reactions after operations, but few have recorded in the literature any details of these reactions. Brunner and Cutler[2] recently reported their observations based on 12 cases of fenestration. They reported having seen during the first twenty-four hours after operation horizontal and rotatory nystagmus of the third degree directed toward the side not operated on. After twenty-four hours they invariably observed horizontal and rotatory nystagmus of the third degree directed to the side of the operation, which disappeared in a few days. In addition, they frequently found slow oscillatory movements of the eyes, synchronous with the pulse and occasionally associated with nystagmus of the head. They compared the labyrinthine findings subsequent to surgical injuries of the semicircular canals with those subsequent to the fenestration operation and noticed in the cases of injury that the spontaneous nystagmus was invariably to the opposite side and thus differs in direction from the nystagmus following fenestration. They attributed this difference to the infection which, they stated, was always associated with the surgical injury, while infection was usually absent in the fenestration. They further stated that if the nystagmus noted after fenestration was directed to the opposite side it was probable that an infection of the fenestra had occurred and that for this reason the operation would not be successful.

Our own observations of the postoperative vertigo and nystagmus of a large number of patients are considerably opposed to those of Brunner and Cutler. Accurate evaluation of the nystagmus occurring during the first twenty-four hours after fenestration is difficult because of the fact that the patient, during this period, is usually extremely drowsy and uncooperative, owing to the rather considerable preoperative sedation. There is difficulty in inducing the patient to arouse sufficiently to fix his attention or vision on a particular object so that nystagmus can be observed. During the analgesic stage, however, during the first eighteen to twenty-four hours a variety of nystagmic movements have been observed. The most constant of these is the slow drifting of the eyes to the side operated on, and in those

2. Brunner, H., and Cutler, M. H.: Labyrinthine Symptoms Subsequent to Fenestration of the Labyrinth, Arch. Otolaryng. **45**:613 (June) 1947.

patients who can arouse sufficiently to fix their eyes there is a horizontal and rotatory nystagmus to the opposite side, the side not operated on. While this is the type of movement most commonly observed, it is by no means always seen. On several occasions the quick component directed to the side operated on has been observed, but this has changed at times during the first day to nystagmus directed toward the side not operated on, and in 2 instances to a distinct downward nystagmus.

No particular determination of the significance of this changing nystagmus which is observed during the first day has been attempted, but it appears to be connected with the prolonged effect of the preoperative sedation and the general anesthesia which these patients undergo at operation. Light nitrous oxide was used throughout the operation in the great majority of these cases of fenestration. When the patient can be aroused sufficiently to cooperate, the nystagmus is nearly always to the side not operated on. The various other eye movements observed are mixed and changeable. In all cases the early nystagmic movements are associated with intense vertigo and severe nausea and vomiting, which is frequently spontaneous through the day and always increased with any disturbing of the patient.

The slow oscillatory movements of the eyes and the nystagmus of the head which have been mentioned by Brunner and Cutler have also been observed by us and are found fairly constant, the degree depending largely on the severity of the labyrinthine reaction.

On the day following the operation almost invariably the nystagmus has been found to be firmly established in the direction toward the ear not operated on. This is the reverse of that reported by Brunner and Cutler. The rapidity and the amplitude of these eye movements vary according to the intensity of the vestibular reaction as registered by vertigo and nausea of the patient. This spontaneous nystagmus usually gradually becomes less marked each day following the operation and in nearly all the cases disappears in from six to twelve days. It is accompanied by vertigo, nausea and frequently vomiting for two or three days following the operation, but in the average case the vertigo and nausea have subsided sufficiently by the end of the third day so that the patient can leave his bed and walk a short distance. Varying degrees of vertigo and sense of unbalance, however, remain with the patient for ten days to a month. During this period following operation, beginning a day after fenestration, the nystagmus, if present at all, is toward the side not operated on.

The fact that the predominate nystagmus is directed to the opposite side for several postoperative days, associated with improvement of the hearing and active fistula response, in our opinion does not mean that infection of the fenestra has occurred, as suggested by Brun-

ner and Cutler. It means, we believe, that the labyrinth is reacting as it should to the traumatic stimulation it has received during the operation. In none of over 200 patients operated on, has there been to our knowledge an actual infection of the labyrinth, as there have been no clinical symptoms suggestive of such an occurrence. The strictly aseptic surgical technic and the phophylactic use of penicillin permit of the manipulation of such delicate structures without fear of an immediate infection. It is true that a rather large percentage of the mastoid cavities become infected at some stage following operation, but in no case has there been observed any symptom referable to the labyrinth that would indicate infection of that structure.

THE FISTULA TEST AFTER FENESTRATION

The postoperative reaction of the labyrinth to the fistula test is of interest and yields a rich field for study and speculation. Some operators place much importance on this response in estimating the success of the operation. Others consider it of little significance as far as the result of the operation is concerned. There are commonly seen two types of results—the negative type, the test having elicited no labyrinthine reaction, and the type in which some nystagmic movement is produced.

The test can be made in the usual way by compressing or aspirating the air of the auditory canal, or it can be made by pressing the fenestrated area or the area adjacent to it with a cotton-tipped applicator. The latter method is commonly employed during the process of cleaning the aural cavity during the postoperative period. It does not as accurately register the response of the vestibular apparatus concerned as does the compression and aspiration method. With the latter method the typically positive result is the production of nystagmus directed to the side of the compression and then, with aspiration, to the opposite side.

Significance of the Positive Result.—When the response to the fistula test is extremely active, it is considered by most operators a favorable sign as far as the hearing result is concerned. It indicates that the labyrinthine function has not been damaged by the operative procedure or by the postoperative reaction, and it shows that there is little or no obstruction to the passage of stimuli such as touch or sound through the fenestra. Both of these are conducive to a good hearing result, and it can be stated that on the whole the more active the response to the fistula test the greater is the postoperative improvement of hearing.

There are, however, exceptions to this rule. At times the postoperative fistula test will elicit no response or weak nystagmus when

the improvement of hearing has been considerable. At other times, as has been observed by many operators, there has been no postoperative improvement of hearing in the presence of active nystagmus. An explanation of some of these phenomena is entirely lacking at this time in many instances.

Significance of the Negative Result.—It has been the experience of all operators to find that in some cases the fistula test produces no labyrinthine response after operation. In probably the majority of cases this negative result is seen in those cases which are considered failures. It means, at least in many instances, that the vestibular mechanism has been damaged and is not capable of functioning. Sometimes this damage and the resultant loss of function are permanent, but frequently the damage is only temporary, and full labyrinthine function is restored after weeks or months.

In a rather large percentage of the patients operated on the immediate effect during the first postoperative day and sometimes for several days is a considerable improvement of hearing coincident with severe vertigo, nausea and nystagmus. In this large percentage the initial improvement and severe labyrinthine reaction are followed by a considerable reduction of the hearing, often well below the preoperative level, along with relief of the vestibular excitation. When the packing is removed from the areas of operation on the third to sixth day after fenestration, it is found that the fistula test produces no response.

In such cases the status of the vestibular apparatus is in doubt. The labyrinth is not dead, as is proved in most cases by the full recovery, in days, weeks or months, of vestibular function. It appears to be in a state of suspended function or of fatigue, and the cochlear function is usually involved in a similar way and to a parallel degree. At times this state of suspended function will be changed quickly—in a matter of hours—to a state of active function with, again, marked improvement of hearing coincident with return of vertigo, nausea, nystagmus and a positive response to the fistula test. At other times the change back to active function is slow and gradual—over a period of days to weeks—with gradually increasing fistula response and gradual improvement of hearing. Again, at times there is never any return of positive reaction to the fistula test, and this frequently means that the hearing result will not be good. However, this is not always so, as some patients will reveal satisfactory improvement of hearing though the fistula test gives no positive result at any time.

Following operation, all degrees of positive reaction to the fistula test may be discovered, from the degree in which the light touching of a cotton-tipped applicator to the fenestrated area may nearly throw

the patient to the floor to the degree in which only a mild response can be elicited by considerable pressure of a cotton-tipped applicator. As mentioned in a foregoing paragraph, in the majority of instances the active response to the fistula test is observed in the patients with the better hearing; but, as also mentioned, there are notable exceptions to this. Adequate explanations of these varying labyrinthine reactions are not available at the present time, and their full significance must await further study and experimentation.

TESTS AVAILABLE FOR DIAGNOSING OTOSCLEROSIS IN AN "OPERABLE" STAGE

There is herewith presented a list of the testing items suggested by this study.

1. Air conduction audiometry.
2. Bone conduction audiometry—good bone conduction is necessary.
3. Fork testing of bone conduction hearing, 512, 1024 and 2048 being used—good bone conduction is necessary.
4. Fatigue test—no evidence of fatigue appears in early otosclerosis.
5. Gellé test—patient's response must be negative.
6. Matching of loudness in the two ears by raising the intensity—impossible in early otosclerosis.
7. Speech-hearing tests showing a loss of hearing over 30 decibels, and articulation tests showing conductive deafness.
8. Caloric test.

In conclusion, we have presented our observations as to audiometric and speech-hearing tests made before and after operation. We have suggested what one may expect to find in the cases in which deafness due to otosclerosis may be dealt with successfully by operation. There may be added a number of hearing tests which we believe to be of considerable value in arriving at a true and appraised diagnosis. We believe that if one will use and obtain experience in making all of the tests, a truer picture of otosclerosis will be obtained, and likewise, that greater discrimination will be exercised in selecting the patient who can be benefited by operation.

SUMMARY

In selecting the patient whom one may treat successfully by fenestration of the labyrinth, it is important that the deafness be carefully evaluated to determine as far as possible how much of the impairment is of the conductive type and how much is of the perceptive type. This can be done, in a large measure, by the following tests:

1. Audiometric tests of air conduction hearing. A careful study of the pure tone audiometric curves of deafened persons is helpful

but not conclusive in estimating the amount of conductive and perceptive loss.

2. Audiometric tests of bone conduction hearing. Although the testing is occasionally confusing and misleading, on the whole it is of great value in estimating the type of deafness if properly done. Measured masking is essential, and it is important to check the airborne sound from a bone conduction receiver.

3. Fork testing of bone conduction hearing. This type of testing can be of great value in estimating cochlear function if the many factors of error are recognized and guarded against.

4. Fatigue test. Auditory fatigue does not show up in catarrhal deafness or early otosclerosis.

5. Gellé test. Here also are numerous factors of error, but the test can be of value in the hands of the experienced tester.

6. Loudness balance test. Matching of the loudnesses in the two ears by raising the intensity is impossible in the stage of otosclerosis considered operable.

7. Speech-hearing tests. In the differentiation of deafness these tests are of value, particularly that involving articulation interpretation.

8. Caloric test. The vestibular response to stimulation such as turning or water douching is helpful in estimating the functional activity of the inner ear mechanism.

In a study of the speech and the pure tone hearing improvement obtained by the fenestration operation, no definite correlation between the two could be discovered. The pure tone hearing may be better as compared with the speech hearing, or the opposite may be found.

A study of the postoperative vertigo and fistula reactions of a large number of patients was made, and several observations were noted. The nystagmus following an operation was almost invariably directed to the nonfenestrated side. The majority of patients operated on showed an active response to the fistula test as performed by compressing the air of the auditory canal or by pressing a cotton-tipped applicator to the area of operation. The active postoperative fistula response is usually associated with the best hearing result. A good hearing result may be obtained in the absence of a positive reaction to the fistula test at any time after operation. There may be no postoperative improvement of hearing in the presence of an active fistula response. Adequate explanations of the varying labyrinthine reactions observed after operation are not available at the present time, and their full significance must await further study and experimentation.

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PHYSIOLOGIC SURGERY OF THE NARES

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RHINOPLASTIC SURGERY has now developed to a point where it must move in the direction of physiology[1] if its functional results are to keep pace with its esthetic attainments. This objective can be realized only through a closer cooperation between the rhinologic surgeon and the physiologist. Unfortunately, the problem of adjusting physiologic theory to practice is difficult. The physiologist who does not directly observe surgical treatment and its results finds himself at a disadvantage in that he is unable to check his theoretic contributions as applied to surgery. The surgeon, on the other hand, has always been reluctant to accept physiologic opinion on the ground that the experimental process in itself interferes in some measure with the phenomena to be observed. He feels that physiology at best presents a rather fragmentary synthesis, which provides only a more or less approximate understanding of any concrete or specific problem.

This gulf must somehow be bridged, for only by the integration of surgery and physiology can both departments advance effectively. The surgeon cannot deny that surgical planning, in its essence, invovles predictions, all of which must be predicated on physiology, and the more complete the physiology the more accurate the prediction. As for the physiologist, the demands of surgery for the solution of unsolved problems will greatly widen the scope of his field. Once the findings of his science are applied to the exigencies of surgery and tested out in the operating room, there will be opportunities for revising his theories with benefit both to him and to the surgeon.

Presented in part as the Birkett Memorial Lecture before the Canadian Medical Association at McGill University, Montreal, Canada, May 9, 1947.

1. (*a*) Fomon, S., and others: Rhinoplastic Analysis, Eye, Ear, Nose & Throat Monthly **24**:19-24 (Jan.) 1945. (*b*) Fomon, S.: Surgery of Injury and Plastic Repair, Baltimore, Williams & Wilkins Company, 1939. (*c*) Fomon, S.; Syracuse, V. R.; Bolotow, N., and Pullen, M.: Plastic Repair of the Deflected Nasal Septum, Arch. Otolaryng. **44**:141-156 (Aug.) 1946.

We shall try to rationalize the surgical procedures from a functional standpoint in an effort to show that rhinoplasty should be considered an integral part of present day rhinology.

CLINICAL ANATOMY

The nares open on the base of the nose and are separated from each other by an anteroposterior septum, the columella. Their downward direction protects the vestibule against the introduction of foreign bodies. They vary from mere slits to voluminous openings and differ in shape and direction with race, heredity, trauma, disease and individual characteristics. In the white race they are elliptic ventrodorsally, while in negroid races they are nearly round, their long axes tending to lie horizontally. It is thought that the direction and the shape of the nostrils bear some relation to the geographic environment. Thus, inhabitants of dry, cold climates have long noses with narrow nostrils, directed downward; these adaptations cause the height of the column of air entering the nose to be raised and its flow retarded, thereby helping to insure that the air will be moistened and warmed. Conversely, those dwelling in hot climates have short noses with large nares, directed forward; thus the height of the curve is lowered and the time during which the air remains in contact with the nasal mucosa is shortened.

The simplest and perhaps the most satisfactory method of analyzing deformities of the nostrils is that dependent on the use of photographs, a face view, a left and a right profile view and a basal view,[1] the photographs being taken half life size and always under similar conditions of lighting and position.

The full face view demonstrates defects in the symmetry, the width and the tip length of the nose (fig. 1*A* and *B*). To analyze—

1. Draw vertical lines through, first, the midintercanthal point and the center of the lip, *a—b;* second, the right inner canthus, parallel to the central line, *c—d;* third, the left inner canthus, parallel to the central line, *e—f*. The nasal components in the two compartments thus formed should be identical. The alae should be tangent to line *c—d* and *e—f*. If they fall within these vertical lines, the interalar width is deficient. If they fall outside these lines, the base is too wide.

2. Draw two horizontal lines through, first, the eyebrows, *g—h,* and, second, the alar-labial junctions, *i—j*. Divide the horizontal space thus formed into quarters by lines *k—l, m—n* and *o—p*. If the height of the alae is correct, line *o—p* will fall tangent to their upper margins.

Note the highlights along the dorsum and the tip for width. The nasal tip should fall slightly below line *i—j* (it is assumed that the photograph was taken with the head in the Frankfort horizontal position). If it lies above this level, the nose is too short; if it lies more than slightly below, the nose is too long. The length of the nose is more accurately determined, however, in profile view.

Fig. 1.—Photographic analysis of the lobule before and after correction: *A* and *B*, face view, *C* and *D*, right profile. (See text.)

The profile view best demonstrates the projection of the tip and the length of the nose.

On the photograph of the right profile (fig. 1 *C* and *D*): 1. Draw line *a*—*b* through the most projecting parts of the forehead and the chin. 2. Draw line *c*—*d* along the most projecting part of the dorsum of the nose, bisecting *a*—*b* at the nasal root, *r*. 3. With a protractor measure angle *b*—*r*—*d*. Ideally, this angle should be 30 to 38 degrees, and the tip should project 2 to 3 degrees beyond the dorsum. 4. Without removing the protractor, mark off a point at

30 to 38 degrees. 5. Draw line *e—f*. The difference between the existing projection and the ideal indicates the amount of reduction necessary.

The photograph of the left profile (fig. 1 *E* and *F*) is used to determine the length of the nose. The most pleasing outline is one in which the nares form an angle of 90 degrees in men and 100 degrees in women with the line of the

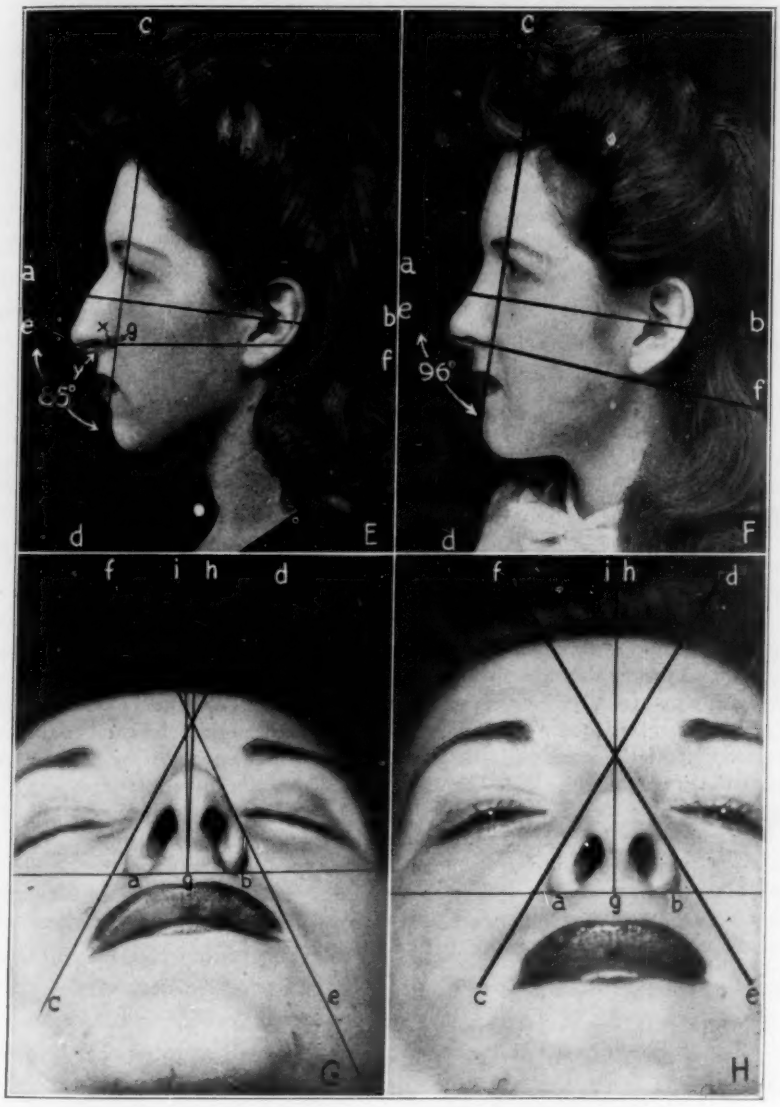

Fig. 1.—*E* and *F*, left profile. *G* and *H*, basal view. (See text.)

upper lip. Owing to the great variability of the shape and the inclination of the lip, the old method of using the lip as a landmark has been discontinued and the following substituted:

1. Draw a line, *a—b*, through two points, one at the center of the lower rim of the orbit and the other at the depression above the tragus (Frankfort

horizontal plane). 2. Draw a line, *c—d*, perpendicular to *a—b* and tangent to the alar-facial junction. Mark the point of intersection *g*. 3. Draw a line, *e—f*, through the long axis of the nostril. Measure angle *e—g—d*. An angle exceeding 100 degrees indicates a short nose; one less than 90 degrees indicates a nose of excessive length.

The profile view also best illustrates the contour of the columella and the character of the nasolabial angle. Mark point *x* where the ala joins the lip. Mark point *y* at the apex of the septolabial angle. Ideally, *x* should lie at a somewhat higher level than *y*. If it is on the same level or below that of *y*, either the columella is retracted or the ala is too low.

Note the contour of the columella below line *e—f*. An undue convexity with exposure of the septal lining constitutes a hanging septum. Inability to see the lower edge of the columella below *e—f* signifies that the columella is retracted or that the alae are excessively wide or low positioned.

The basal view photograph (fig. 1 *G* and *H*) demonstrates: The shape and symmetry of the nostrils, the width of the columella, the thickness and contour of the alae, and the breadth of the lobule and the base. To analyze—

1. Draw a line, *a—b*, through the points where the alae join the lip. 2. Draw lines *c—d* and *e—f* along the walls of the alae. 3. Draw line *g—h*, beginning at a point midway between *a* and *b* and passing through the intersection of *c—d* and *e—f*. 4. Draw a line perpendicular to *a—b*. If *g—i* does not coincide with *g—h*, it indicates a deviation of the lower cartilaginous vault, the degree being determined by angle *i—g—h*.

Observe the nostrils. Ideally, they should be roughly elliptic and converge toward the tip. Note their shape—whether slitlike, comma shaped, round, or irregular—their convergence and their symmetry.

Observe the columella. From its apex to the philtrum it should equal the height of the upper lip. Its width should not exceed that of each nostril. Note the flare of the mesial crura. They sometimes project into the nares and interfere with respiration. Observe the alae. They may be delicate or puffy, round or flat, triangular, square or almond shaped. Their normal height equals one-quarter that of the nose, and the interalar distance is equal to the distance between the inner canthi. Excessive thickness of the alae may interfere with respiration. If they are thin and nonresistant, they may collapse during inspiration.

Examine the lobule, which comprises the area ventral to the nostrils. It should be roughly triangular, with a rounded apex, but may be thick, thin or cleft. Its width across the apexes of the nostrils is approximately three-quarters the width of the base of the nose at the level of the alar-facial junction. The ventrodorsal length is one-third that of the columella.

PHYSIOLOGY

The following account of nasal function is quoted freely from Proetz.[2]

To render the inspired air acceptable to the lung, the nares play a fundamental role, since they regulate the volume, the pressure and the distribution of the inspired air. If they are deformed, there result disturbances related to all three factors.

2. Proetz, A. W.: Applied Physiology of the Nose, St. Louis, Annals Publishing Co., 1941.

Volume.—The amount of air entering the nose is largely controlled by the size of the nares, and inasmuch as the effective working of the mucosa is dependent on adequate ventilation, nares too large will interfere with ventilation by admitting an excessive amount of air, and nares too small will prevent the introduction of the requisite amount.

Adequate Pressure.—The flow of gases in the nasal chambers is dependent on pressure fluctuation set up by the thoracic excursion in combination with the constriction of the nares. The nares, being invariably somewhat smaller than the choanae, govern to a large extent the pressure differentials. Mink[3] stated that the degree of negative pressure determines the height of the inspiratory arch (fig. 2). According to Bernoulli's principle,[4] even minor variations in cross section result in pressure changes which cause considerable physiologic disturbance:

1. Normally the nares, being slightly smaller than the choanae, produce a negative pressure of approximately minus 6 mm. of water on inspiration and a positive pressure of plus 6 mm. of water on expiration.

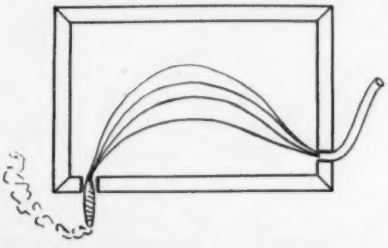

Fig. 2.—Diagram of a glass box with a cigar at the inlet and a pipe at the outlet, to demonstrate the effect of negative pressure on the height of the inspiratory curve. (Mink's[3] illustration, modified.)

According to Proetz, as long as the ostiums remain at all patent, the pressure changes in the sinuses are identical with those in the nose. Thus, deformity of the nares must interfere with drainage of the sinuses and its consequences. Insufficient pressure fails to make a respiratory impression and thus is unable to influence the reflexes which inaugurate the mechanism of respiration. Too great pressure has the same effect because of the obtunding influence of drying and irritation.

2. With narrowing of the nostrils, the thoracic bellows forces against the constricted nares and accentuates the negative and the positive pressure (fig. 3).

3. When the nares are large, the thoracic bellows forces against the constricted choanae, and nasal pressures are quickly equalized through the large nares and only slight fluctuations take place.

3. Mink, P. J.: Physiologie der oberen Luftwege, Leipzig, F. C. W. Vogel, 1920.

4. Duff, A. W.: Principles of Physics, ed. 8, Philadelphia, The Blakiston Company, 1946.

Distribution.—According to Proetz, it is necessary to nasal health that all portions of the nasal fossae be accessible to air currents, that they be accessible in proper proportion and that the stream of air proceed without too much eddying. Otherwise, a chain of symptoms arises marked by anosmia, headache, metaplasia and infection.

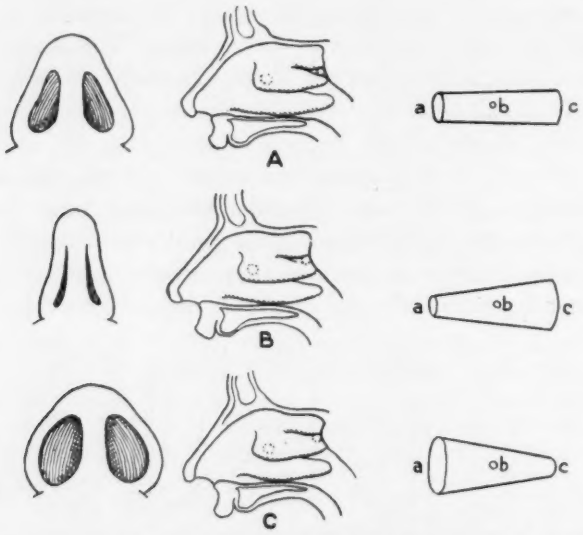

Fig. 3.—Control of pressure differentials by the nares. *A*, normal nares: (*a*) nares; (*b*) sinuses; (*c*) choanae. (See text.) *B*, constricted nares, accentuating negative and positive pressures. *C*, abnormally large nares, diminishing negative and positive pressures. (See text.) (Proetz's[2] illustration redrawn.)

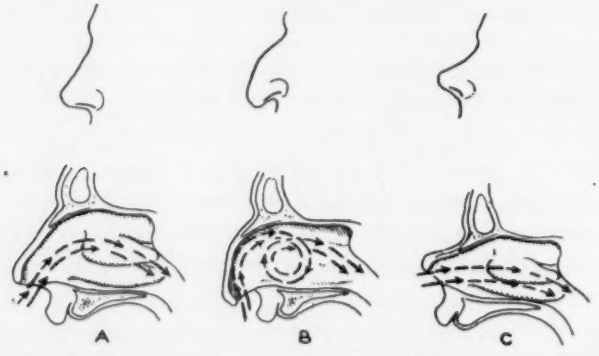

Fig. 4.—Inspiratory current: *A*, normal parabolic curve. *B*, impingement of the inspiratory current on the dorsum and formation of eddies in the case of dependent tip. *C*, flattening of the arch and more direct course of the current in the case of unduly elevated nares.

The inspiratory current normally takes a parabolic curve (fig. 4*A*) since, the nares being dependent and the piriform opening being at a somewhat higher level, the current must necessarily arch from inlet to exit, and, as previously mentioned, the height of the current is governed

by the negative pressure. Without this arrangement, the directional effect of the nares would be lost and the currents redistributed. In the case of unduly dependent nares, the inspired air impinges on the dorsum (fig. 4*B*), and the irritation of the eddy thus created playing on the area of the anterior ethmoidal nerve may occasion reflex disturbances. Clinically, there is a localized drying out of the mucosa, also a piling up and drying of the nasal mucus, which by its irritation causes local inflammation, discomfort and pain. The bacteria in this favorable medium flourish and have a tendency to penetrate the mucosa. If, on the other hand, the nares are unduly elevated, the arch is flattened and the current

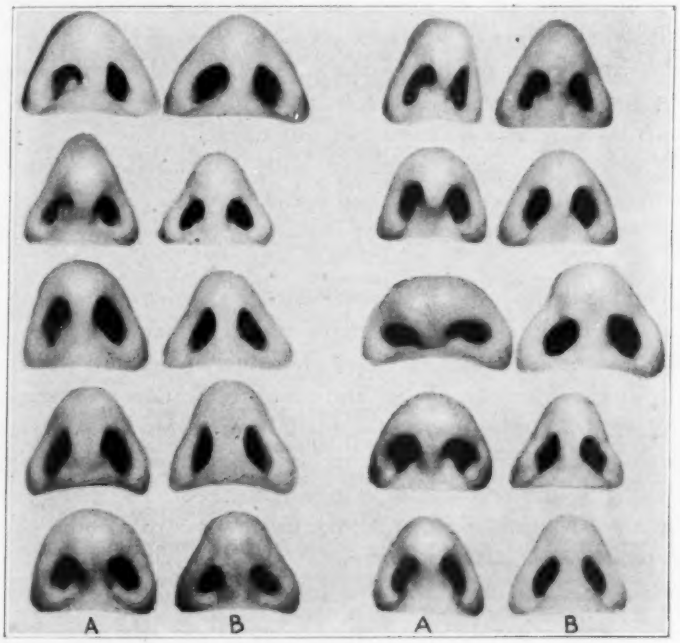

Fig. 5.—*A*, types of nares interfering with function. *B*, restoration of function by reconstruction.

takes a more direct course (fig. 4 *C*). This also leads to drying because it interferes with the precipitation of water on expiration.

PATHOLOGY

The nostrils may be faulty in shape—i.e., too wide, too narrow, elongated (slitlike or collapsed), shortened or asymmetric; in size—i.e., too large or too small; in their plane of directions, i.e., tilted upward or downward; or in axis—i.e., oblique or transverse. Any or all of these faults may interfere with the volume, the pressure or the distribution of the respiratory air.

No classification can embrace the infinite variety of deformities of the nares (fig. 5). But with a clear understanding of the individual abnormalities which may affect their boundaries—i.e., ventral or tip, dorsal or lip, mesial or columella, and lateral or alae—the various combinations of pathologic defects can be easily integrated and the surgical correction readily planned.

Ventral Boundary (Nasal Tip).—The nostrils are bounded ventrally by the tip, the normal contour of which is due to the graceful rounding of the angles of the lower lateral cartilages. Tip deformities causing nasal abnormalities include the following:

1. Wide tip usually associated with a widening of the angles of the lower lateral cartilages or to an excess of soft tissue.

2. Narrow tip. It usually speaks for a narrowing of the ventral pole of the nares and is caused either by a narrowing of the angles of the lower cartilages or by a deficiency of soft tissue.

3. Twisted tip. It is associated with asymmetric nostrils and is caused by a dislocation of the caudal end of the septum, or by hypertrophy, buckling or overlapping of the angles of the lower lateral cartilages.

4. Pendulous tip. It is characterized by a downward tilting of the nares in which the slant of the nares forms an angle of less than 90 degrees with the philtrum. We have encountered noses in which the tip reached almost to the upper lip, and nasal respiration was possible only when the end of the nose was raised with the fingers. Patients with this deformity tell of holding the tip in an elevated position during sleep by attaching it with adhesive tape to the forehead. The condition is due either to an elongation of the middle or the lower cartilaginous vault or to a retraction of the septolabial angle.

5. Retroussé tip. This is associated with an upward tilting of the nares such that they form an angle of more than 100 degrees with the philtrum. Anatomically, this is due to a short septum and short upper lateral cartilages, an upward tilting of the lower lateral cartilages or an enlarged nasal spine which widens or obliterates the nasolabial angle.

6. Prominent tip. This is accompanied by elongated nostrils. It is due either to abnormal length of the lower lateral cartilages in an anteroposterior direction or to their being dislocated forward, or to an excess of soft tissue.

7. Recessed tip causing small or negroid nares. It is due to abnormal shortness of the lower lateral cartilages in the anteroposterior direction or their backward dislocation, or by shortness of the columella.

Lateral Boundaries (Alae).—The lateral boundaries of the nares comprise the alae, whose conformation is largely controlled by the lateral crura of the lower lateral cartilages. They are subject to a wide

variety of deformities: 1. As to curvature, they may be convex and produce the typical rounded (flaring) nostril of the negroid type; or concave, producing narrow, or pinched, nostrils. 2. As to dimensions, they may be abnormally long, producing voluminous nostrils, or abnormally short, the size of the nostrils being proportionately smaller. 3. As to position, they may be elevated or depressed, either as a matter of heredity or as a result of cicatricial contraction following trauma or disease. 4. As to width, they may be so thick as to encroach on the nares or so thin as to increase their size.

Mesial Boundaries (*Columella*).—The mesial boundaries of the nares are formed by the columella, the contour of which depends largely on the flare of the mesial crura and their soft tissue investment.

The columella may be too wide, producing narrow nostrils; too narrow, producing wide nostrils; too short, giving rise to small nostrils whose axes run from side to side; too long, and then usually associated with slitlike nostrils. The condition known as "hanging septum" occurs when the mesial crura are abnormally wide, or when the septum is too long, forcing the columella below its normal level, or when an abnormally long columella deforms the base of the nose by its redundancy. Retraction of the columella occurs when the septum has been dislocated posteriorly from the spines of the maxillas or has been resected to relieve obstruction, or it may occur as a sequel to excessive shortening of the nose in a rhinoplastic operation. An oblique columella results in asymmetric nostrils. Sometimes it is caused by cicatricial contraction; or the agent may be a septum displaced from its position in the median line. An enlarged nasal spine and buckled mesial crura also may be responsible for columellar deformities.

Dorsal Boundary (*Nasolabial Junction*).—The nasolabial junction may be too wide or too narrow, each affecting the size of the dorsal pole of the nares. It may be recessed, as in the harelip deformity, or may protrude, as in alveolar prognathism.

MANAGEMENT

Seldom can defects of the nares be attributed to a single factor. They are usually the result of a combination of abnormalities about the boundaries of the nostrils. For example, the nares may be abnormally large because the columella is long and thin, the alae are long and thin or the nasolabial junction is too wide, or because of a combination of these abnormal findings. A tilting downward of the nares may be due to an elongation of the septum and the upper lateral cartilages, a retraction of the septolabial angle or a downward tilting of the lower lateral cartilages. A diagnosis of the causative factors is therefore extremely important if a good functional result is to be expected.

Obviously, the operative procedures for the correction of the deformities should be described from the standpoint of those affecting the volume, the pressure and the distribution of the respiratory current. This, however, would entail much repetition, because of the overlapping of the many causative factors. They will therefore be described in relation to the structures bounding the orifices of the nares. A knowledge of the basic procedures about to be described, together with a careful analysis, will enable one to improvise the proper treatment for any combination of structural faults.

A Procedure Preliminary to All Corrections of the Nares: Exposure of the Lower Lateral Cartilages (fig. 6).—This procedure is essential to the correction of the majority of the abnormalities described and is accompliished in the following manner:

Anesthesia is obtained with a 1.5 per cent procaine hydrochloride solution containing 1:1,000 epinephrine hydrochloride solution, 10 drops to the ounce. With a Bard-Parker no. 11 knife an incision is made along the "plica nasi" (an anatomic shelf formed by the lower margin of the upper lateral cartilage when

Fig. 6.—Exposure of the lower lateral cartilages. (See text.)

the tip of the nose is elevated). The aponeurosis connecting the upper and lower lateral cartilages is incised, the incision beginning at the center of the plica and extending ventrally to the septum. The procedure is repeated on th opposite side. A button end knife is passed from one incision to the other, and the membranous septum is cut through, the knife being held close to the caudal margin of the septum. An incision is now made in the vestibule just below the caudal (inferior) margin of the lower lateral cartilage and the procedure repeated on the opposite side. A scissors is introduced into the incision and brought out through the initial intercartilaginous incision, the instrument lying on the plane between the external skin and the cartilage. The blades are now opened and the overlying tissues separated from the cartilage. With a dural hook placed at exactly the angle of junction between the lateral and mesial crura, the cartilage is drawn out of the vestibule and divided at the angle.[1b] From here the modeling will depend on the objective to be achieved.

VENTRAL BOUNDARIES OF THE NARES (CORRECTION ON THE TIP).—The following technics were used.

Wide Tip.—If the angles of the lower lateral cartilages are at fault, the following procedure is resorted to: The cartilages are delivered and the angles cut

through. Strips of cartilage are resected from the cephalic portions (superior margins) of the lateral crura, their size depending on the degree of deformity. If the vestibules are of normal size, the resection is limited to the cartilage. If, however, they are too large, strips of vestibular skin are removed as well. The overlap between the upper and lower lateral cartilages having been lessened by the aforementioned resection, the lateral bulbousness is eliminated. Four or 5 mm. of vestibular skin is then removed from the cut ends of the lateral crura to obviate the burying of the epithelium (fig 7). The deepithelized portions are brought together over the mesial crura in the form of an inverted V. This approximation serves not only to narrow the tip but to give it a more natural appearance by projecting it slightly beyond the dorsal line.

If the width is due to an excess of soft tissue, or if the cartilages are thin, soft and lacking in resiliency, modeling of the cartilages will not suffice; excisions of tissue must be resorted to. The choice of procedure will depend on the size of the nares. If they are of normal size, two elongated ellipses of skin and subcutaneous tissue are excised from the area just above the nares, and the mar-

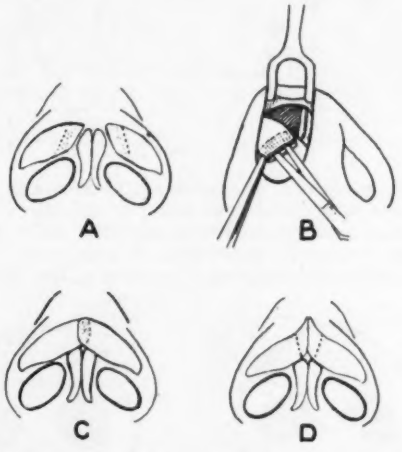

Fig. 7.—Narrowing the nasal tip: *A*, angles cut through. *B*, vestibular skin lining removed from the cut end of the lateral crus for a distance of 4 to 5 mm. *C*, deepithelized surfaces of the lateral crura overlapped. *D*, deepithelized surfaces brought together over the mesial crura in the form of an inverted V.

gins of the wound are approximated with atraumatic sutures (fig. 8*A* and *B*). If the orifices are too large, the semilunar sections are cut in such a manner as to include a portion of the apical rim (fig. 8*C* and *D*). Approximation of the raw edges will reduce not only the width of the lobule but also that of the nares. If the nares are too small, openings of normal size are outlined in methylene blue on the base of the lobule (fig. 9). An incision is carried into the nares through the center of the area outlined. The subcutaneous tissue beneath the two small flaps thus outlined is dissected out and the skin rolled inward to cover the raw surfaces. The flaps are fixed in place by gauze packing of the nostrils.

Pendulous Tip.—If the upper cartilaginous vault is at fault, it constitutes a part of the long nose deformity and the tip must be elevated. To create room for such an adjustment, triangles of cartilage must be resected from the septum and the upper lateral cartilages. Obviously, the bases of the triangles must lie on the dorsum and the apexes be directed toward the nasal spine. The technic can be found in any standard textbook. Briefly, the soft tissues of the nose are elevated,

and the membranous septum is divided in the usual manner. The upper lateral cartilages are freed from their attachments to the septum for a distance equal to the base of the triangle to be removed. The tip of the nose is raised with the finger until the nose assumes the desired length. The triangle of septum which now protrudes through the nostril represents excess cartilage and is removed with a pair of Mayo scissors. The space thus created is obliterated by fixing the columella to

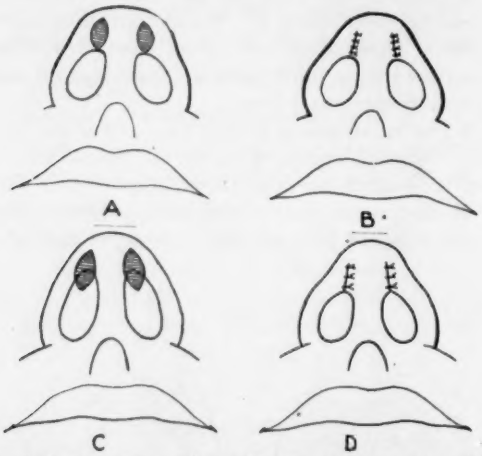

Fig. 8.—*A* and *B*, correction of a wide tip in a case in which the nares are normal and the excessive width is due to excess of soft tissue: *A*, elliptic portions of skin and subcutaneous tissue excised from the area above the nares; B, wound margins approximated. *C* and *D*, correction of wide nares (orifices too large): *C*, semilunar sections removed, including a portion of the apical rim; *D*, margins approximated.

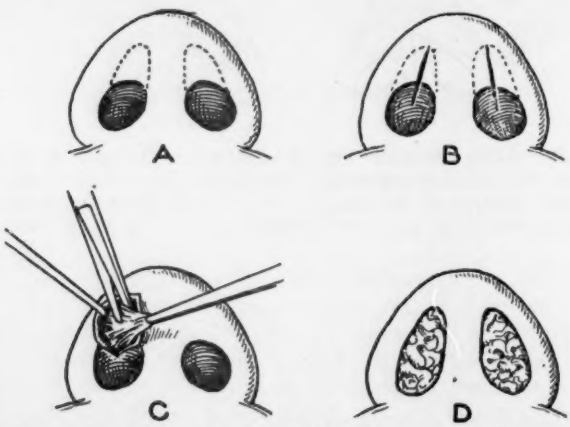

Fig. 9.—Correction of a wide tip with small nares: *A*, normal openings for the nares outlined in methylene blue. *B*, incision carried into the nares through the center of the area outlined. *C*, subcutaneous tissue excised. *D*, skin rolled in to cover the raw surface and held in place with petrolatum-treated gauze packs.

the septal cartilage with two strong silk sutures. This automatically causes the lower lateral cartilages to tilt upward and thus restores the normal nasolabial angle. The upper lateral cartilages, which are now too long for the shortened nose, protrude into the vestibule and are excised (fig. 10).

If the dependent tip is due to abnormal width (cephalocaudal length) of the lower lateral cartilages, room is created for tilting the tip upward by excising strips from the cephalic (superior) margins of the lower lateral cartilages (fig. 11*A*).

If the lower lateral cartilages not only are too wide but also take a downward course, they create the impression, after the columella has been attached to the septum, of an overhanging tip with a retracted columella. To avoid this defect, the caudal margin of the lateral crus near the tip is rounded off. Occasion-

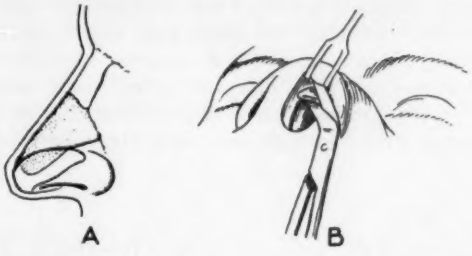

Fig. 10.—Excision of protruding portions of the upper lateral cartilages: *A*, upper lateral cartilages too long for a shortened nose. *B*, excision of the protruding portion.

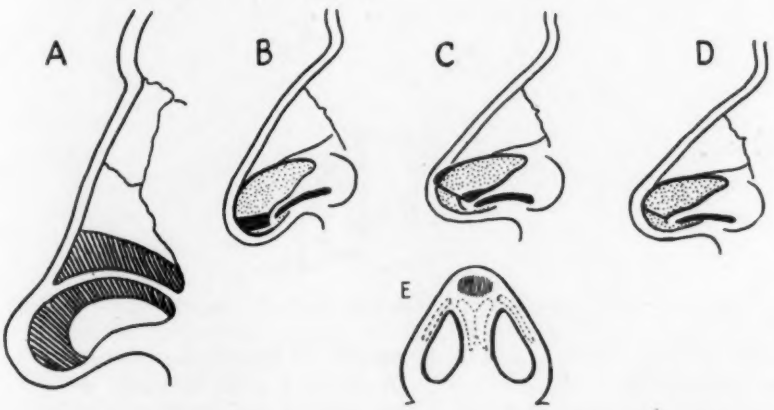

Fig. 11.—*A*, correction of a dependent tip, the deformity of which is due to abnormal size of the upper and/or the lower lateral cartilages: The shaded areas indicate the location of the cartilage to be resected to make room for elevation.

B, *C* and *D*, correction of a dependent tip, the deformity of which is due to downward displacement of the lower lateral cartilages. In *B* the shaded area indicates the section of cartilage to be removed. In *C* elevation brings the two unequal margins together. Cross-hatched areas indicate the cartilage to be trimmed. *D*, result of trimming.

E, modeled cartilage inserted above the mesial crura to restore the contour of the tip.

ally it is necessary to treat the cephalic margin in like manner (fig. 11*B*, *C* and *D*). When the dome of the lower lateral cartilages is flat and thick and will not straighten out when made to override the mesial crura, the cartilage is best sacrificed and replaced with a modeled button of cartilage (fig. 11*E*).

Prominent Tip.—A projecting tip may be due to one of several causes. The treatment obviously will depend on the anatomic findings. If the cause is an unduly high septum, resection of the ventral border of the septum and adjacent parts of the upper lateral cartilages will allow the lower lateral cartilages to seat themselves in a more recessed position. If the abnormality is due to a forward dislocation of the cartilages, these are severed from the upper cartilaginous vault, set backward and attached to the septum in their corrected positions. If abnormal length of the cartilages is the underlying factor, the angles are delivered, cut through and resections made from the lateral and mesial crura, of a size sufficient to produce the normal projection when they fall together.

With all these deformities the soft tissue must also be carried backward, and this is accomplished by freeing the base of the nose from the alveolus, drawing it backward and maintaining it by means of a perforating suture[5]: One needle of a double-armed suture enters the skin of the columella and is pushed through to emerge from one of the columellar walls. It is then carried through the dorso-

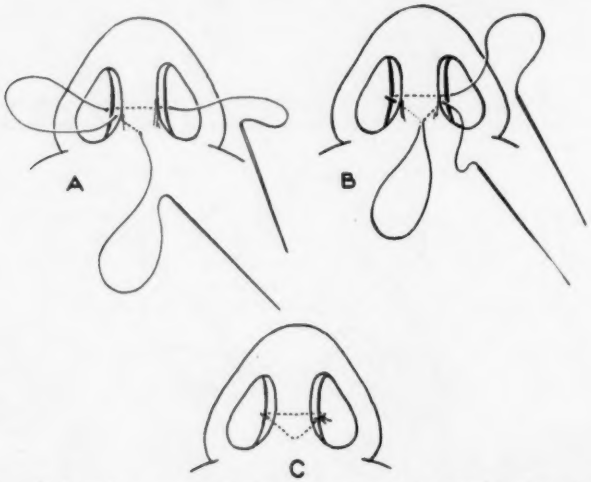

Fig. 12.—Perforating suture designed to carry the tip backward. (See text.)

caudal (posterior-inferior) part of the septum and brought out through the naris. The other needle is carried through the original point of entry, brought out through the other columellar wall, and the ends are tied (fig. 12). It is a difficult suture to remove. Therefore, it is advisable to insert a piece of silk in the loop before it is tied.

In this deformity there is frequently a marked disproportion between the length of the philtrum and the base of the nose, the lip being abnormally short. A more balanced relationship between these two structures can be brought about by placing the perforating suture in front of the septolabial angle, i.e., in the dorsal part of the columella. When the ends are tied, the suture will draw the columellar tissues into the lip and thus lengthen it.

Recessed Tip.—The recessed tip, like the projecting tip, may be due to one of several causes, and here, too, the success of the operative procedure will naturally depend on a correct diagnosis.

5. Daley, J.: Role of Columellar and Septocolumellar Sutures in Rhinoplasty, Arch. Otolaryng. **45**:178-190 (Feb.) 1947.

(1) If the deformity is due to backward dislocation of the lower lateral cartilages, the lower cartilaginous vault is separated from the upper cartilaginous vault. The lobule is brought forward and maintained in position by two sutures passed between the columella and the septum.

(2) If the deformity is due to insufficient development of the cartilages, the overlying soft tissues are freed and a button of modeled cartilage is placed above the angles of the cartilages to bring the tip forward to the proper projection (fig. 11*E*).

(3) If the deformity is due to flaccidity of the structures forming the columella—in this case the columella is soft and bent on itself—the rigidity is restored by inserting a batten of cartilage into a columellar pocket made for the purpose.

(4) If the columella is short, the following procedure is employed (fig. 13): The alae are separated from the cheeks. Through these incisions the base of the nose is separated from the upper part of each maxilla, care being taken to hug

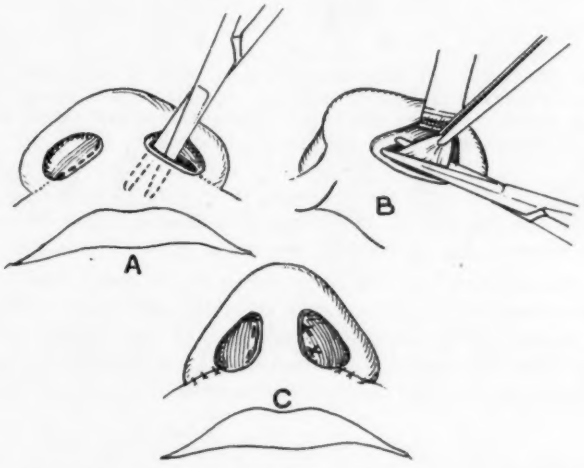

Fig. 13.—Lengthening of the columella: *A*, ala separated from the cheek, and base of the nose separated from the maxilla. *B*, subcutaneous tissue removed. *C*, mattress suture inserted to increase columellar length at the expense of labial width. Alar incision closed.

the bone with the scissors to avoid perforating into the gingivolabial sulcus. Subcutaneous tissue is removed, and a mattress suture is passed through the septolabial angle which, when tied, will increase the columellar length at the expense of labial width. The columella is reattached to the septum in a more forward position, and the alae are then sutured back in place with atraumatic sutures.

LATERAL BOUNDARIES OF THE NARES (CORRECTION OF THE ALAE.)—Descriptions of the technics used follow.

Long Alae.—This defect gives rise to elongated nostrils, which may be either slitlike or voluminous. In either case the technic for their correction is as follows (fig. 14): The alae are pressed in to locate the alar-facial junctions and then separated from the cheeks. The alar rim is forced beneath the mesial margin of the wound until the proper configuration is obtained. While the ala is held in

this position, a mark is made on it along the line of contact, and the excess tissue is removed with a Bard-Parker knife. The wound margins are coapted with atraumatic sutures, which are left in place three or four days. The incisions having been made along natural creases, the remaining scars are barely perceptible.

Convex Alae.—Convex alae are treated as long alae, with this difference in determining the amount of tissue to be removed (fig. 15): The ala is pressed

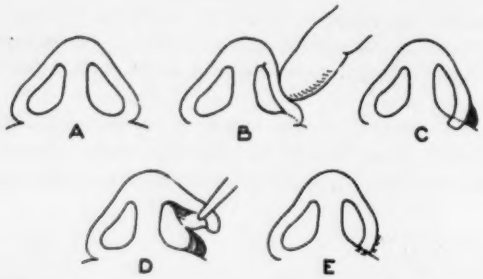

Fig. 14.—Reduction of long alae: *A,* long alae. *B,* ala pressed against the cheek to determine the alar-facial junction. *C,* ala forced beneath the medial margin of the wound to secure the proper configuration, and excess tissue marked out. *D,* excess tissue removed. *E,* reduced ala reattached.

inward with the finger until the convexity disappears. The line of contact between the ala and the base of the nostril is marked out, the semilunar portion of skin lying between this line and the alar-facial groove is removed.

Thick Alae.—Thick alae are corrected in the following manner (fig. 16): The alae are separated from the cheeks along the alar-facial grooves. The cut edge shows a fatty pad between the vestibular and the external skin, definitely demarcated. This fatty wedge is removed with a no. 11 Bard-Parker knife, and the skin surfaces are brought together with a suture, the ends of which are tied

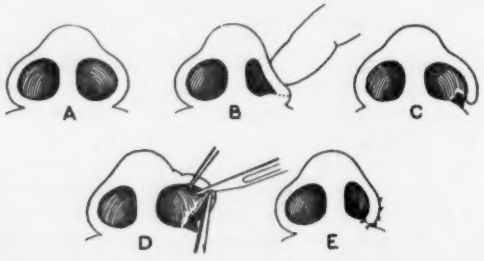

Fig. 15.—Reduction of convex alae: *A,* deformity. *B,* ala pressed in with the finger until the convexity disappears. *C,* ala cut through at the point of angulation. *D,* excess tissue removed. *E,* wound margins coapted with atraumatic sutures.

inside the vestibule. The suture is so passed that it is returned through the same skin opening through which it was brought out, thus obviating any danger of surface scarring.

Subcutaneous tissue may be removed similarly from the wound margins on the cheek and the lip. This will serve to equalize the raw surfaces which are to be approximated.

Bulging of Lateral Crura into the Vestibule.—Here the lateral crura of the lower lateral cartilages bow inward so as to encroach on the lumens of the nares. The bowing is usually manifested externally by a deepening of the alar groove, which gives the nose a pinched appearance.

An incision is made in the vestibule along the caudal margin of the bowed cartilage, and the soft structures above the cartilage are elevated. The cartilage is

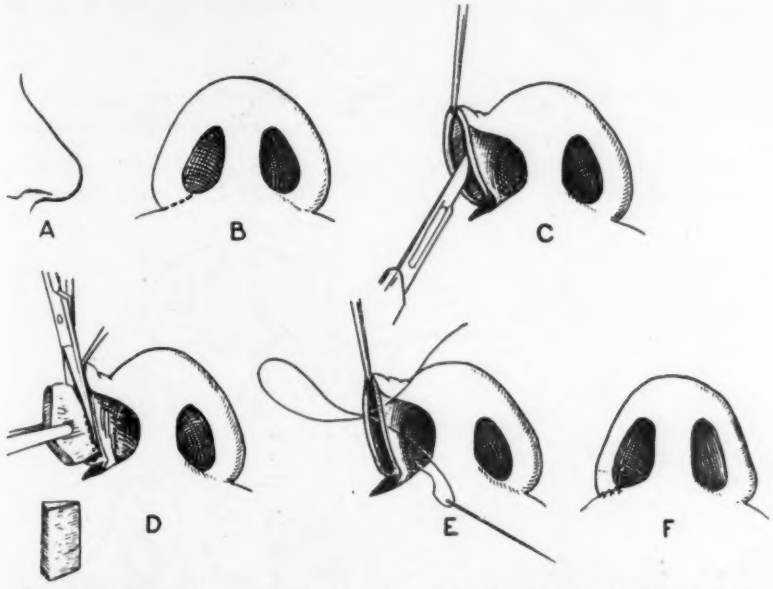

Fig. 16.—Reduction of thick ala: *A* and *B*, alae separated from the cheeks along the alar-facial grooves. *C*, sharp dissection of the fatty pad between the skin layers. *D*, resection of the fatty wedge. *E*, edges of the skin brought together with a silk suture so passed that the loop disappears beneath the cutaneous surface. *F*, alae reattached.

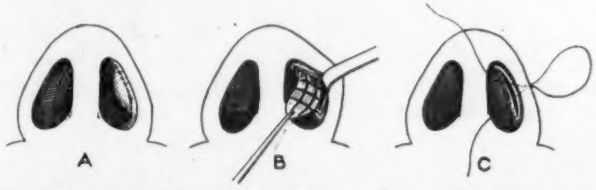

Fig. 17.—Correction of inward bulging of the lateral crura: *A*, incision made in the vestibule along the caudal margin of the bowed cartilage. *B*, lateral crus delivered and cross-hatched. *C*, vestibular skin attached to the outside skin by a perforating suture.

cross-hatched or parts are excised, and the vestibular skin is fixed to the outside skin by a perforating suture tied on the vestibule surface (fig. 17).

Flat Alae.—Flat alae constitute a part of the harelip deformity and are corrected as follows (fig. 18): The osseous and upper cartilaginous vaults are reconstructed according to the technic employed in a typical rhinoplasty. The lower lateral cartilages are completely mobilized. The affected ala is separated from the cheek along the alar-facial groove and is thinned out in the manner described under

"Thick Alae." A perforating suture is passed as previously described, and in such a manner as to take up the redundant vestibular skin. A pocket is made in the columella, and a batten of cartilage is introduced. The two sides are twisted between the thumb and the forefinger so as to swing the short side upward to the level of the long side. The parts are maintained in the corrected position by two mattress sutures passing through the batten. If the base of the nose is depressed, it is elevated at this time by a cartilage batten, introduced through the wound created by elevation of the ala. Additional grafts may be necessary

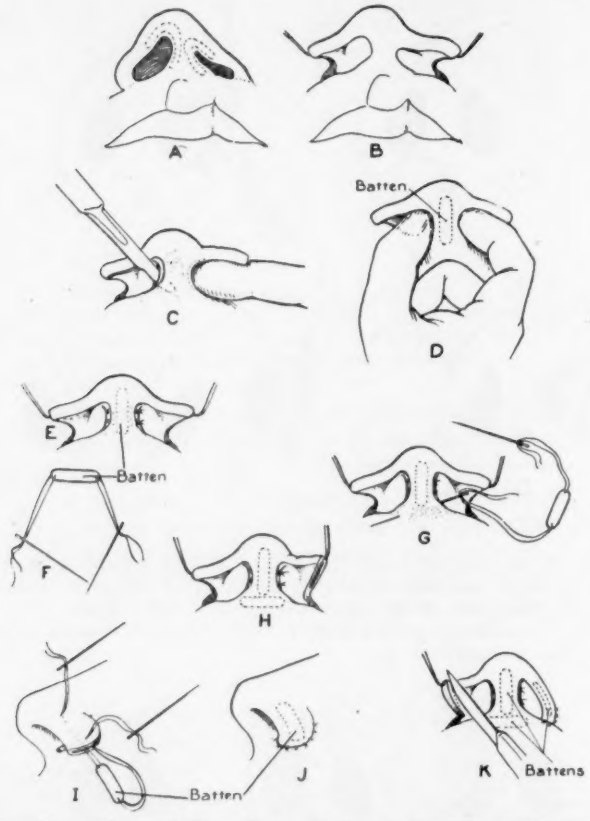

Fig. 18.—Correction of flat alae: *A,* lower lateral cartilages completely mobilized. (Refer to figure 5.) *B,* alae separated from the cheeks along the alar-facial grooves and thinned out as in figure 15. *C,* pocket made in the columella. *D,* cartilage batten put in place and the short side swung to the level of the long side by twisting motion. *E,* parts maintained in the correct position by two mattress sutures passed through the batten. *F,* pilot suture passed through batten. *G,* pilot sutures passed through lip. *H,* graft in place. *I* and *J,* alar margins stiffened by additional grafts. *K,* normal ala made to match the corrected side.

to stiffen the alar margins. Finally, the ala is rotated inward and reattached to the lip with atraumatic sutures. The normal ala is reconstructed to match the corrected one.

Abnormal Elevation of the Lateral Crura.—Here the lateral crura, instead of running parallel to the nares, tilt upward, the fault being characterized externally by a facet on the alar rim.

The lower lateral cartilages are delivered (fig. 19). The lateral crus is freed from its outer and vestibular skin investments. A suture is passed through the terminal end of the cartilage. The free end of the suture thread is passed through the eye of the needle. The needle, now holding both ends of the suture thread, is passed into the pocket created by the liberation of the cartilage and then is brought out on the surface at the point where the lateral crus is to be relocated. Traction is made, drawing the crus into the desired position, and an atraumatic suture is passed through the vestibular skin and the cartilage to hold it in place.

MEDIAL BOUNDARIES OF THE NARES (CORRECTION OF THE COLUMELLA).—The methods used were as follows.

Thick Columella.—If the columella is too thick because of excessive soft tissue or because of too much flaring of the extremities of the mesial crura, in-

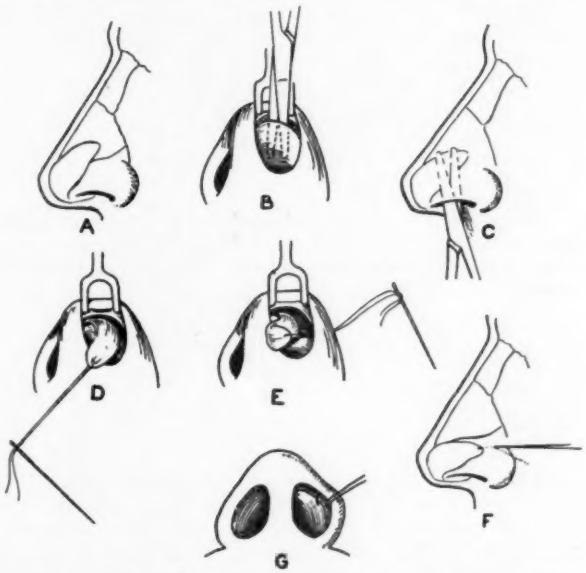

Fig. 19.—Correction of abnormal elevation of the lateral crura: *A*, position of the lateral crus. *B*, vestibular skin separated from the lateral crus. *C*, outer skin separated. *D*, lateral end of cartilage drawn out and suture passed. The needle contains both ends of suture thread. *E*, needle passed into the pocket created by liberation of cartilage and the suture brought out on the surface where the lateral crus is to be relocated. *F*, crus drawn down to the desired position by traction on suture. *G*, cartilage held in place by atraumatic suture.

cisions are made along the cephalic margins of the mesial crura and the excess subcutaneous tissue and cartilage are excised (fig. 20). The mesial crura are then sutured together with black silk. This will result in an improved airway, for the size of the nares has been increased.

Long Columella.—Although we have performed rhinoplasty several thousand times, we have never once had to excise the redundancy of a long columella externally. When the lower lateral cartilages have been completely mobilized and reduced, the skin adapts itself readily. Any excess may be thrown into the lip by resorting to the perforating suture previously described (fig. 12).

Short Columella.—The elongation of a short columella has already been described under "Recessed Tip" (fig. 13).

Retracted Columella.—This deformity is usually due to a dislocation of the caudal end of the septum, to a sacrifice of the caudal portion in a case of obstructing septum, or to excessive shortening of the septum in a rhinoplastic operation. The method of correction will depend on the cause and on the presence or the absence of a membranous septum.

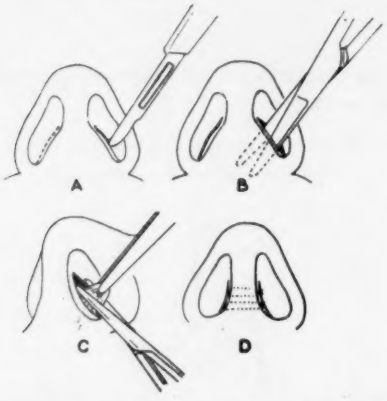

Fig. 20.—Correction of a thick columella: *A*, incisions made along the cephalic margins of the mesial crura. *B*, columellar skin separated from the underlying structures, *C*, excess tissue removed, *D*, mesial crura united with mattress sutures of black silk.

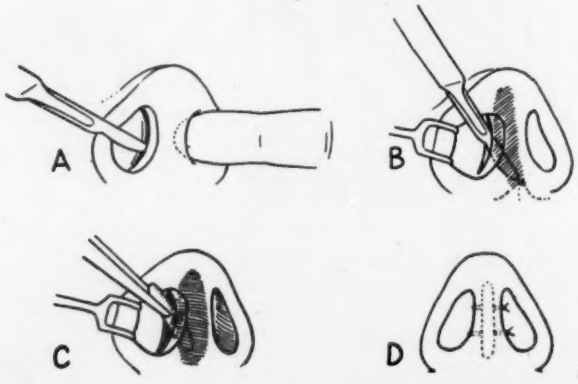

Fig. 21.—Correction of retracted columella: *A*, membranous septum incised, the index finger protecting the opposite wall against perforation. *B*, bed prepared in columella for reception of a graft. *C*, columellar graft inserted into the prepared pocket. *D*, graft held in place by sutures passed behind it.

When it is due to a displaced septum, the retraction disappears as soon as the septum has been restored to its sagittal position. When excessive shortening is the cause, a unilateral incision is made in the membranous septum, through which a bed is created and a batten of cartilage introduced (fig. 21). In cases in which there is insufficient membrane left to enclose the batten, membrane is borrowed from the septum as follows (fig. 22.): The membrane on one side

of the septal cartilage is incised 5 to 6 mm. cephalad and parallel to the caudal margin of the septum. The membrane is freed from above down. That on the opposite side is similarly treated. Between the two layers of the newly made membranous septum a batten of cartilage is implanted and is fixed in place by two mattress sutures passed through the displaced membranes.

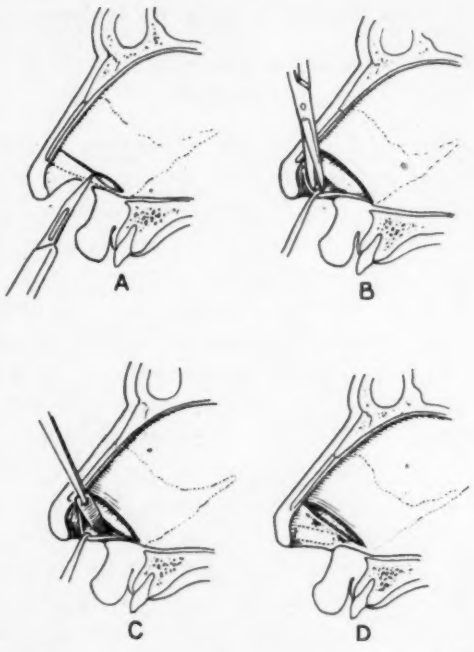

Fig. 22.—Correction of retracted columella with deficient membranous septum: *A*, incision made in the mucoperichondrium above the caudal margin of the septum. *B*, membrane freed below the incision. *C*, batten of cartilage implanted in the pocket. *D*, cartilage graft fixed in place with two mattress sutures.

SUMMARY

An attempt is made to rationalize the surgical correction of abnormalities of the nares from a functional standpoint and to show that rhinoplasty should be considered an integral part of present day rhinology. The clinical anatomy, physiology and pathology of the nares are reviewed.

The corrective surgical procedures described in this article have been developed in our clinic. They will help restore normal volume, pressure and distribution of respiratory air. Frequently the procedures serve as adjuncts to septal surgery in the restoration of function. It is felt that many technically perfect submucous resections of the septum have failed to restore normal nasal respiration because the necessary surgical correction of the nares was neglected.

ELONGATED STYLOID PROCESS

Further Observations and a New Syndrome

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DURHAM, N. C.

IN 1937 I[1] presented the first case of clinical symptoms due to an elongated styloid process observed in the clinic of the division of otolaryngology of the department of surgery of Duke University School of Medicine. At that time I reported 2 cases in which there had been surgical shortening of the elongated process and 2 cases in which there had not been an operation of this type. In 1940 a series of 43 private cases with a diagnosis of elongated styloid process was reported from our clinic by Fritz,[2] which included the 4 previously mentioned cases. From 1940 to December 1946 we have observed 211 additional cases on the private service at Duke Hospital, making a total of 254 cases, an average of 35 each year on the private service alone. There has been a total of 44 operations on these private patients, for shortening of the styloid processes. The condition is diagnosed with less frequency in the public dispensary than in the private clinic, owing to the failure to perform routine palpation of the pharynx and to the fact that the number of Negro patients having tonsillectomy, with the ensuing symptoms, is small. The Negro race probably has proportionately more elongated styloid processes than the white race, but such styloid processes are more often found in anatomic dissecting rooms.

In view of the frequency of the severity of the clinical symptoms my colleagues and I consider the subject of elongated styloid process worthy of still further reports. Even now the disease is not recognized or treated by many otolaryngologists. This is not surprising, because nine of ten of the newer editions of textbooks in the field of otolaryn-

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1. Eagle, W. W.: Elongated Styloid Processes: Report of Two Cases, Arch. Otolaryng. **25**:584 (May) 1937.

2. Fritz, M.: Elongated Styloid Process: Cause of Obscure Throat Symptoms, Arch. Otolaryng. **31**:911 (June) 1940.

gology do not even include the disease. These facts have doubled our efforts to accentuate and stimulate the interest of otolaryngologists in the subject. In addition to the usual symptom complex, which was previously described, another syndrome is presented, which we believe may be traced directly to an elongated styloid process.

Begging indulgence, I should like to describe my first clinical experience with an elongated styloid process. In 1935, we saw once again a patient whom we had seen over a five year period at fairly frequent intervals, who had complained of pain in the left side of her throat and pain in the left ear, with the pains being more severe on swallowing. The tonsils had been removed eight years prior to her first visit, or thirteen years previously. She always had the sensation that her throat had never healed after the tonsillar operation. Needless to say, she had returned many times to the specialist who had removed her tonsils and had also visited numerous other otolaryngologists. She was considered to be a neurotic person by each of us who had observed her, because at no time were any of us ever able to see anything wrong in her throat. Finally, in 1935, thinking "she may have a foreign body, such as a toothbrush bristle or a fishbone, in the mucosa of the pharynx or the hypopharynx, which can not be seen, even with a laryngeal mirror," I decided to palpate the pharynx. To my surprise I did palpate something firm in the left tonsillar fossa, which was not palpable likewise in the right tonsillar fossa. The left fossa was extremely tender on palpation, and the palpation reproduced the pain in the pharynx and the left ear of which she had been complaining. A skull was studied, and, with the mandible wide open, it was evident that only a styloid process could have been palpated in the left tonsillar fossa. During my studies of anatomy I had seen and dissected a styloid process, but throughout the intervening years I had lost all memory of the styloid process, as there had never been any clinical significance of the styloid process in my practice. The literature was then reviewed, and I was amazed to find that little had been published on clinical symptoms referable to the elongated styloid process. Most of the articles were found in anatomic journals, and in these articles the condition was considered as an anatomic curiosity. The patient previously referred to was then told of her probable condition. She submitted to a surgical shortening of the left styloid process, and immediately there was complete alleviation of all her symptoms.

It is not necessary or important that I review here the historical background of the clinical recognition of the elongated styloid process. This was reviewed in my previous presentation.[1] I do wish to emphasize, however, that patients having symptoms as in the case just described have in each instance undergone tonsillectomy, and the

symptoms immediately followed the operation. The other syndrome which I shall describe is atypical and may occur in patients who have or have not had their tonsils removed.

Patients are now classified into two groups—first, those patients exhibiting the typical syndrome as in the case just cited; second, those patients having pain beginning in the neck, which follows the distribution of the carotid artery, either the external or the internal branch, usually the external branch. The carotid artery syndrome, I believe, is due to the elongated process that points either medially, causing pressure and some obstruction of the internal carotid artery, or laterally, thereby impinging on and impairing the circulation in the external carotid artery. In either instance, pain follows the distribution of the artery.

SYMPTOMS

Typical Syndrome.—The characteristic symptoms are: a constant pain or a nagging dull ache in the pharynx, presenting the sensation that the throat did not heal after tonsillectomy; a pain frequently referred to the ear, indicating irritation of the vagus nerve; increased salivation; hesitancy and difficulty in swallowing; gagging; a sensation of a foreign body—even cotton—in the pharynx. These symptoms may be wrongly diagnosed as due to glossopharyngeal neuralgia, but when analyzed they do not fit in with true glossopharyngeal neuralgia, since in that disease one deals with neuralgia of extreme severity, usually lancinating and of momentary duration, but recurrent and frequently stimulated by intake of hot or cold stimuli, such as food or drink, and sometimes stimulated by even the slightest movements of the tongue. The pains due to elongated styloid processes are constantly nagging but rarely severe.

The group of cases presenting the typical syndrome also includes all cases in which there is distortion of nerve function involving the sensory and motor fibers of the fifth, seventh, ninth and tenth cranial nerves. The sensation of taste may be quite distorted. A spasm or contraction of the constrictor muscles of the esophagus and the pharynx may be observed. One patient having spasm of the constrictor muscles was caused much embarrassment each time he attempted to eat. Shortening of his elongated processes allowed him to eat normally.

Patients having typical symptoms due to an elongated process, I repeat, are those who have undergone tonsillectomy and whose symptoms arise during the post-tonsillectomy convalescence. Scar tissue formation stretching the nerve endings is thought to be the cause of the symptoms. The ninth cranial nerve is most often affected, with occasional fifth nerve, and rare seventh and tenth nerve involvements. The tautness of the pharyngeal mucosa over the process can easily

stimulate these nerve endings as movements of the pharynx occur. In 1 case I noted the pharyngeal mucosa suspended as if on a tent pole, and when the patient gagged, the mucosa of the pharynx visibly glided over the tip of the process, each movement causing pain. The typical syndrome is found when the elongated process points straight forward or medially—the usual direction.

I do not infer that all elongated styloid processes become symptomatic, for many an elongated process is found on routine palpation of the pharynx when the patient is complaining of no local symptoms whatever. Certainly, operation is not recommended unless the elongated process is causing severe symptoms.

Carotid Artery Syndrome.—In August 1946 I had the first data to substantiate an idea which I had entertained for six years that an elongated styloid process might produce pains along the carotid artery distribution. Since 1940 I had encountered patients, probably 12 in all, who had complained of pain beginning in the neck at a point about opposite the tonsillar fossa, which extended upward over the side of the head in a pattern conforming to the distribution of the external or the internal carotid artery. I had made notes that probably a carotid artery was involved in these cases in some way, but I had no definite evidence of this.

Mrs. S., aged 67, was first seen in May 1946, complaining of a pain of many years' duration throughout the left side of her head, beginning in the neck, and the pain was in a perfect pattern of the external carotid artery distribution. She was found to have elongated styloid processes, but the left one only was tender on palpation beneath the tonsils, which were still present. Since all other examinations gave entirely negative results, the consultants decided that the left styloid process should be shortened, and in August 1946 the patient returned for the operation. After the left tonsil was removed, the left styloid process was found deviated or hooked at the tip and firmly embedded against the external carotid artery, restricting its lumen. She had an uneventful convalescence and to the present date has been relieved of her symptoms. (It is of interest to note that she has a sister who also has a bilateral elongated process, which in her case is asymptomatic.)

This case is the first and only one of the group of cases of suspected carotid artery syndrome in which the patient underwent operation, and thus allowed substantiation of the existence of the carotid artery syndrome. There were other operative cases in which the carotid artery was very superficial, owing to its being elevated laterally by the elongated process, but there was little impingement on the arterial lumen in any of these cases. From the single case cited, it is impossible to conclude too decisively that the other patients in this group had the same basic condition—that is, obstruction to the arterial function and symptoms of pressure on the sympathetic nerves of the arterial walls. The findings at operation are more conclusive than the result of roentgenologic examination and other diagnostic procedures.

Further operations must be done in such cases to establish the syndrome definitely. The posterior-anterior roentgenogram is essential to demonstrate deviations at the tip of an elongated process, but even so they do not demonstrate whether the tip of the process actually cuts into the path of a carotid artery. Occasionally an elongated process resembles a ram's horn (fig. 1), curving on its own axis, which is easily shown in the lateral roentgenogram. In any case suspected to be one of the carotid artery syndrome posterior-anterior, as well as lateral, roentgenograms should be made.

Tinnitus is occasionally complained of by patients having the carotid artery syndrome. One patient had annoying tinnitus, which was almost unbearable when the styloid processes were palpated. This

Fig. 1.—Ram's horn type of elongated styloid process.

aggravation of tinnitus may seem difficult to explain, since the basilar artery, which is a continuation of the vertebral artery and not of the carotid artery, is the only artery supplying the cochlea. The case is similar to one of vascular tinnitus in which there is no elongation of the styloid process, in that the tinnitus is transmitted to the cochlea through the bone.

A pain in one or both sides of the neck may cause a person to consult a physician, and the examiner may find only a superficial and pulsating carotid artery. Frequently this may be the internal carotid

artery, for the textbooks of anatomy relate that just above the bifurcation the internal carotid artery is more external than the external carotid artery itself. In some cases it is superficial and painful on palpation because it is elevated to its position by an underlying elongated and outward-deviating styloid process. (The tip of a process of normal length lies between the two arteries.) In addition, the patient may have increased his symptoms by continually bruising that portion of the cervical region by palpation. A prominent vessel may be mistaken for a cervical lymph node, especially if it is tender. A cervical node overlying a carotid vessel is always quite painful because of the sympathetic sensory nerve supply in the wall of the carotid vessel.

I believe that an elongated styloid process which fits so tightly against a carotid artery as to impair its circulation may cause the carotid artery syndrome. I now wonder and even suspect that many patients are treated for migraine, histamine sensitivity, temporal artery and other vascular headache, or atypical headaches and neuralgias, when in reality they may have an elongated styloid process. It is conceivable that even a styloid process of normal length may cause symptoms if there are anatomic malarrangements and variations in the cervical region. These anatomic variations and the relations of structures in the styloid area, the function of the three styloid muscles and other anatomic facts are under investigation for a future report.

DIAGNOSIS

The diagnosis of elongated styloid process should be facilitated by the patient's history. It is confirmed by the palpating finger. When the tonsils have been removed, there is no question of the diagnosis if a firm bony resistance is met in the tonsillar fossa, especially at the upper pole of the fossa (fig. 2). If a styloid process is palpable, it is always elongated. The normal length of the styloid process is 1 inch (2.5 cm.), and the normal process can never be palpated, since it lies just that deeply in the parapharyngeal musculature. When a styloid process is symptomatic, the patient will wince from pain as the process is palpated, and he will state that the pain is the same as that of which he has complained. The elongated process may be palpated through and beneath some tonsils. Roentgenograms are taken merely for confirmation of the diagnosis but are later used as a guide, and are of considerable aid, in the surgical shortening of the process.

The actual length of the elongated process is determined on the lateral roentgenogram, which also allows the surgeon to determine just how much of the process should be removed at the operation (fig. 3). The posterior-anterior view (fig. 4*A*) shows any deviation of

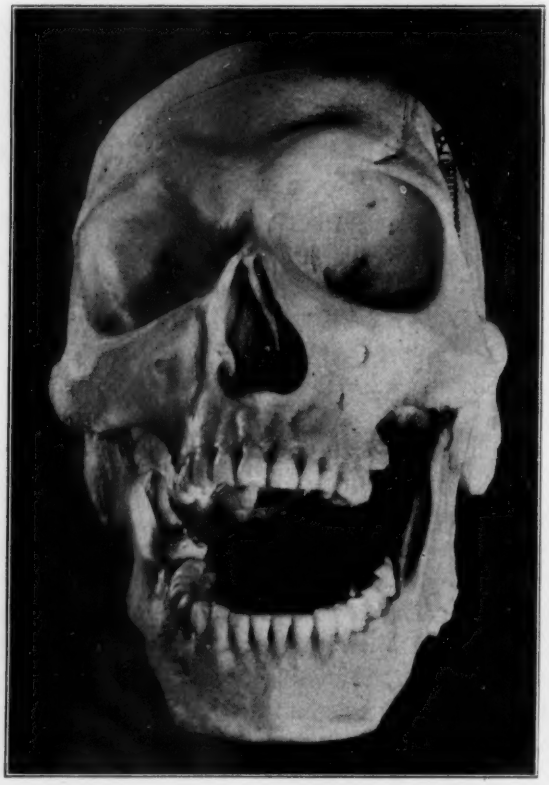


Fig. 2.—The ease with which the elongated styloid process of the right side can be palpated is demonstrated here. The proximity of the styloid process to the mandible is also to be noted.

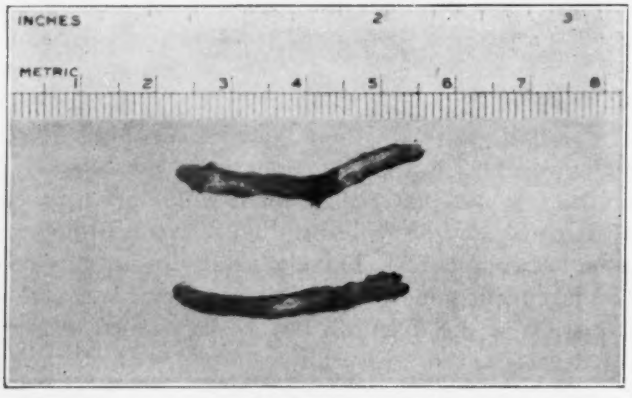


Fig. 3.—Removed elongated styloid processes. Note the curvature of the upper specimen. A styloid process of this type may project against a carotid artery.

the entire process, including the tip, which cannot be recognized in the lateral view.

TREATMENT

The treatment of the elongated styloid process is entirely surgical. Simple digital fracture of the process, experience has shown, does not alleviate the symptoms. Uniformly good results have been obtained by the surgical shortening of the process in all our patients.

The operation for shortening the elongated styloid process is difficult to the extent that one does not have the visualization that is obtained in other operative fields, and the operator must depend to a certain extent on use of the index finger, as well as that of instruments, for the dissection.

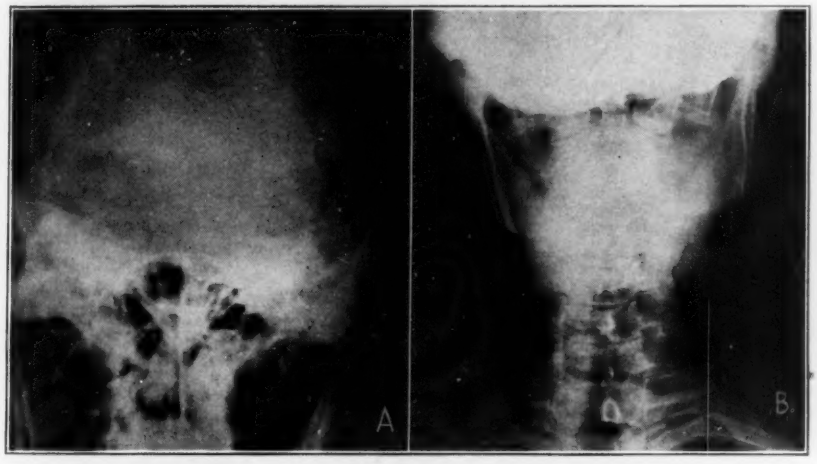


Fig. 4.—In *A* an elongated styloid process is seen on each side. These processes are quite large also.
B shows an elongated styloid process on the left side and a process of normal length on the right side.

Figure 5 illustrates the operation. An incision is made in the tonsillar fossa over the tip of the palpable elongated process. The incision is spread with scissors or a clamp until the tip of the styloid process is palpable through the incision. The muscles attached to the medial surface of the process are stripped by the finger. A curved instrument, such as a submucous resection elevator, is used to free the muscles from the lateral and posterior portion of the process. Very little force may be used during the dissection, lest the process be fractured prematurely. After the muscular stripping, the styloid process stands out as a white, ivory-appearing rod, usually the caliber of a safety match. The stylohyoid ligament at the tip of the process is easily visualized and palpated during the dissection and is now severed

with scissors, unless it is calcified. This maneuver provides easier access for the finger and the instruments to strip any remaining muscular attachments from the posterior and lateral surface of the process. The length of the portion which is to be removed has been determined prior to operation by study of roentgenograms. The actual fracturing of the portion to be removed is accomplished with the Hajek forceps. A curved clamp must have already grasped the distal end of the process to prevent its retraction into the muscles should there be attachments remaining. In removing the fragment by means of this clamp, one must exercise care with regard to the external and internal carotid arteries and the facial nerve, which emerges from the skull posteriorly

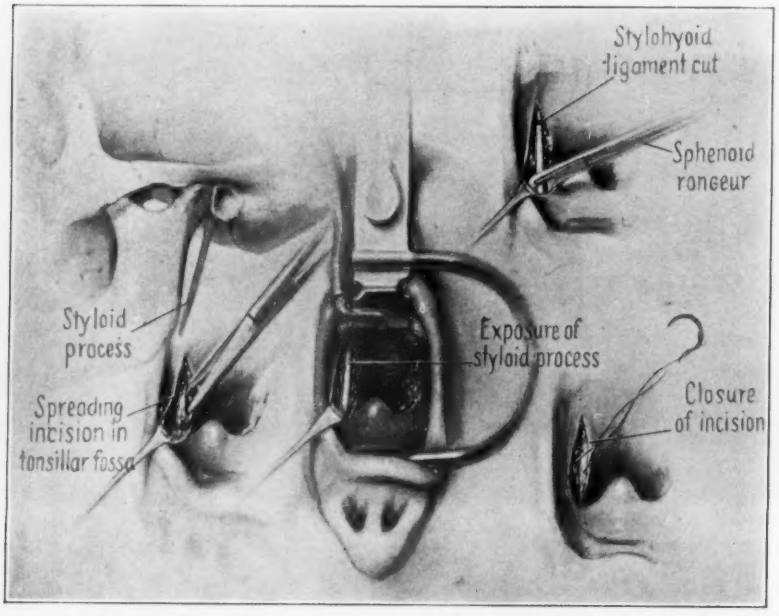


Fig. 5.—The various stages of the intraoral shortening of the elongated styloid process. General anesthesia and the Crowe-Davis mouth gag are used. Sulfanilamide is left in the wound before closure.

to the base of the process. The styloid stump is then palpated to determine whether it is too long, and usually it is not. Sulfanilamide is then placed in the wound, and the incision of the tonsillar fossa is closed with no 0 plain catgut sutures. There is usually no bleeding of significance during the operation, and in our experience hemorrhage has never occurred postoperatively. Edema of the tonsillar fossa is usually mild, and the convalescence is unusually much easier than that following tonsillectomy. This apparently is due to the complete closure of the wound and the action of the sulfanilamide inserted in the wound, preventing infection.

Loeser and Cardwell[3] in 1942 devised another operation to shorten the elongated styloid process. They make a V-shaped incision beneath the mandible, extending it down the anterior border of the sternocleidomastoid muscle. Their operation requires that the contents of the carotid sheath and the other deep structures of the neck be pulled forward in order to expose the styloid process. We are not convinced that the external route is the easier or the more rational method of surgical procedure, and we do not see the necessity of the visible scar which will be present postoperatively in the neck. Furthermore, the fact that the palpating finger locates the process in the tonsillar fossa makes it seem logical that the surgical procedure is an intraoral one.

COMPLICATIONS

There are no complications as such of cases of symptomatic elongated styloid process other than the continuance and increased severity of symptoms. Postoperatively the complications are minimal and actually negligible. There had been no complications of any kind in any of our operative cases until November 1946, when 1 patient had tremendous edema of the neck on each side following bilateral operation. The edema lasted five days and was considered as due to sulfonamide drug sensitivity, the sulfanilamide having been left in the wound. There was no intrapharyngeal swelling in the patient. As a matter of fact, no more edema is noted after surgical shortening of an elongated styloid process than after any other type of pharyngeal surgery. Respiratory distress has never been noted. Patients are usually able to eat well by the third day and are allowed to leave the hospital on the third or fourth postoperative day. In no instance has the facial nerve or a carotid artery been injured.

RESULTS

When the elongated styloid process is surgically shortened, the results are uniformly excellent and permanent. The elongated portion of the styloid process must be fully shortened, with removal of the fragment, to alleviate all symptoms. There have been 2 cases in which the fragment was pulled back into the musculature by some muscles still attached to the process, and the fragments were never recovered; one of the patients still had mild symptoms one year after the operation when last heard from, and the other was operated on only a few months ago, so far without report of trouble. In 1 case in which the styloid process projected parallel to the cervical spine, fracturing was

3. Loeser, L. H., and Cardwell, E. P.: Elongated Styloid Process: Cause of Glossopharyngeal Neuralgia, Arch. Otolaryng. **36**:198 (Aug.) 1942.

done only because of the odd position of the process. The patient still had mild symptoms two years later when last heard from.

SUMMARY

The established syndrome of an elongated styloid process, the syndrome which is typical, is reviewed. An atypical syndrome, referable to involvement of the external or the internal carotid artery, is presented for consideration. The symptoms, the diagnosis and the treatment of both syndromes are discussed. The intraoral surgical technic for shortening the elongated styloid process is described. Complications are reported as minimal, and the intraoral surgical approach is thought to be ideal for excellent and permanent results.

LYMPHOID HYPERPLASIA OF THE EPIPHARYNX

A New Method for Control

S. EUGENE DALTON, M.D.
VENTNOR CITY, N. J.

THE NEW FEATURE of this method is the added emphasis of thyroid dysfunction and infection of the paranasal sinuses as etiologic factors in the production of hyperplasia and hypertrophy of lymphoid tissue in the epipharynx. It follows that the correction of these pathologic conditions will help alleviate the abnormal growth of this tissue and the sequelae of such growth.

ANATOMY

Functionally, the epipharynx is an annex to the respiratory portion of the nasal cavities. In contradistinction to the rest of the pharynx, it normally remains open at all times. It is bounded anteriorly by the choanae; the posterior wall is nearly vertical, extending upward to blend with the vault, and is bounded by the spinal column. The roof lies under the body of the sphenoid bone and extends to the superior margins of the orifices of the eustachian tube. The floor is the upper surface of the soft palate. Each side wall is formed by the orifice of the eustachian tube, the fossa of Rosenmuller and torus tubarius. The epipharyngeal space is lined by mucosa, which contains ciliated columnar epithelium and the lymphoid layer. Beneath this is the muscular layer expect in the fornix. Here the mucosa is attached to the bone only by a thin hard connective layer. Between the mucomuscular layer and the prevertebral cervical fascia is the so-called retrovisceral space, which contains compound tubular mucoserous lymph glands. The middle gland is present during childhood, but the two lateral glands at the level of the atlas are not present until later in life. Three groups of epipharyngeal pouches may be noted in the photographs of this area. Four pouches with slitlike apertures are situated beneath the lower borders of the inferior turbinates. The

The photographs in natural color accompanying this article were taken from the "kodachrome" motion picture of the epipharynx presented before the American Academy of Ophthalmology and Otolaryngology, in Chicago, in October 1945, by S. Eugene Dalton, M.D.

third group, triangular in shape, occupies the central part of the postepipharyngeal wall. These pouches are vascular and harbor numerous bacteria, as revealed by cultures. Infections are readily absorbed from them into the blood and lymphatic vessels. The bony vault of the pharynx is the site of the pharyngeal tonsils. Here also is the site where hyperplasia of the lymphoid tissue occurs in many cases after the adenoid tissue has been removed.

The mucosa of the epipharynx is continuous with that of the eustachian tube. However, there are some structural changes of importance. The epithelium is of the ciliated columnar variety, while the tunica propria is divided into three well defined layers: namely, the basement membrane, a lymphoid and a glandular layer. The lymphoid layer varies in different parts of the tube, being very thin in bony portions and well developed at the pharyngeal end. Sometimes the lymphoid layer at this end is referred to as the tubal tonsil. It contains numerous ducts from glands in the glandular area and has a rich vascular supply of the lymphatic network.

The important relations of the lateral aspects of the cartilaginous portion of the eustachian tube are those with the tensor palati muscle and the mandibular division of the trigeminus nerve, the middle meningeal artery, the otic ganglion and the medial (internal) pterygoid muscle. Beneath the mucosa of the eustachian tube lies a most efficient lymphatic drainage system.

LYMPHOID HYPERPLASIA AND EFFECT OF IRRADIATION

Briefly the chief alterations noted on histologic examination of hypertrophied lymphoid tissue prior to irradiation are hyperplasia and chronic inflammation.

A, four pouches with slitlike apertures that occur beneath each inferior turbinate.

B, third group of pouches, triangular in shape, located in the central part of the postepipharyngeal wall.

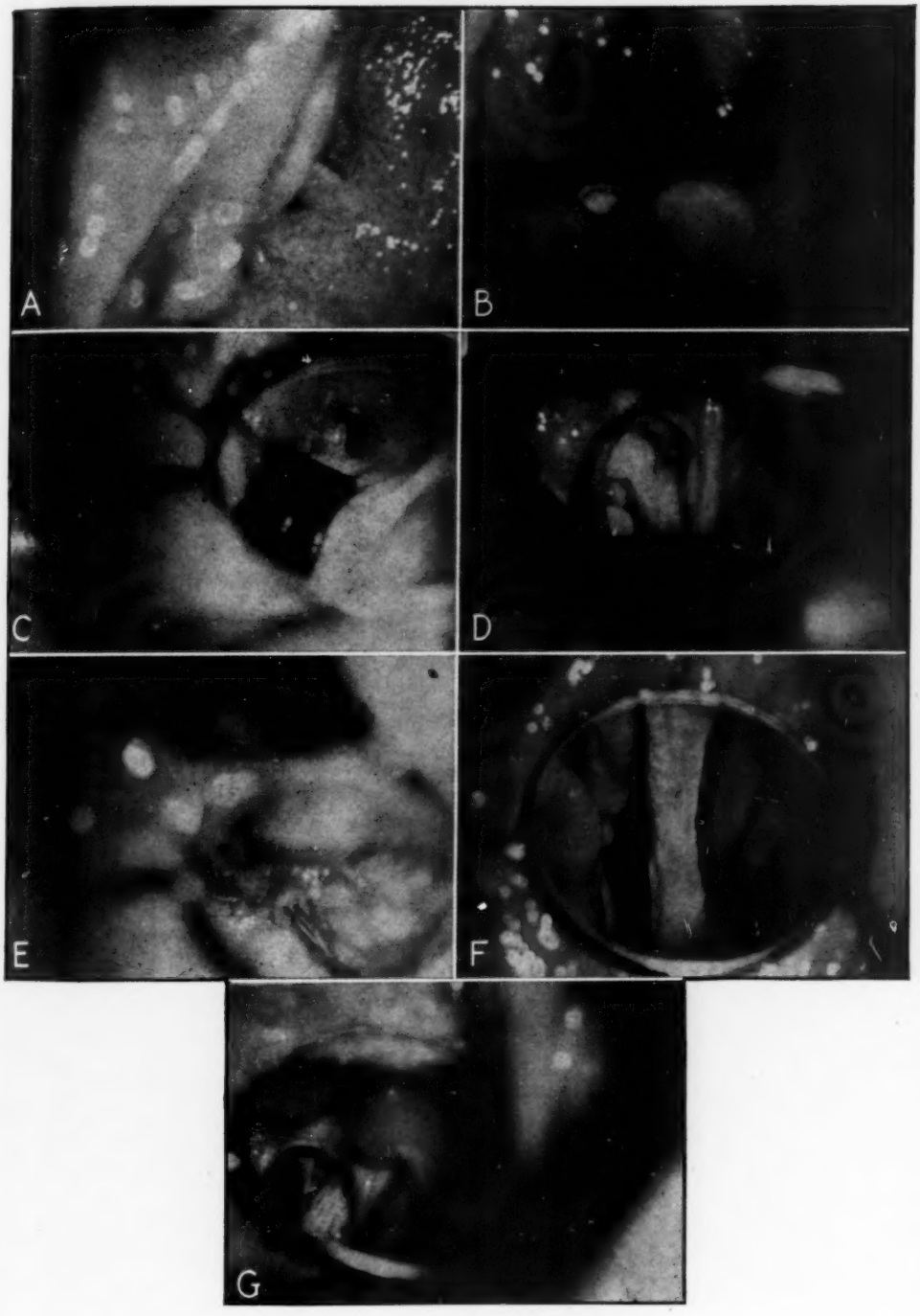
C, epipharynx that had been almost completely obstructed by hypertrophied and hyperplastic lymphoid tissue for the past fourteen years. The patient was a young woman suffering from suppurative sinusitis and hyperthyroidism. Her basal metabolic rate was plus 22.

D, epipharynx of same young woman eighteen months after treatment and operation. The basal metabolic rate was plus 4.

E, hypertrophied lymphoid tissue causing partial obstruction of the epipharynx of a man aged 21, who had suppurative sinusitis and hypothroidism. His basal metabolic rate was minus 23. The tonsils and adenoids had been removed at 2, 8 and 15 years of age.

F, epipharynx of same patient three years after cure. The posterior border of the septum and the middle and inferior turbinates are shown by oral mirror photography.

G, in the torus tubarius, the orifice of the eustachian tube, a part of the inferior turbinate and the posterior border of the septum are shown.



(See legend on page 642)

The rapid proliferation of the cells of the nodules appears to be followed by an increase in the number of polyblasts. The amount of edema within the nodules varies but is most conspicuous in those removed from allergic patients. Dilatation of lymph channels occurs beyond the nodules while lymph channels within and around the nodules appear to be largely obstructed and frequently obliterated. Obliteration is caused by fibroblastic activity and proliferation of the endothelial cells. Although there is initial hyperplasia of the adjacent glandular structure, atrophy ensues if chronic inflammatory activity is prolonged.

Lymphocytes are regarded as the most radiosensitive of the cells of the body. They show high radiosensitivity and a great ability to regenerate after small doses of radiation.[1] Similarly the lymph tissues throughout the body demonstrate high radiosensitivity and great powers of recovery after small doses. Suberythema amounts of radiation cause degeneration of lymphocytic nuclei and breaking up of germinal centers within a few hours. The destruction of lymphocytes continues for days after such irradiation and is accompanied by considerable macrophage activity. Higher doses result in a more rapid and profound destruction of lymphocytes, with decrease in the size of nodes due to destruction of most of their contained cells. Repair takes place by collapse of the nodes with connective tissue replacement.[1a] If the connective tissue is extensive, the node becomes less resistant to infection.

The mechanism whereby abnormal lymphoid tissue produces profound changes in the middle and perhaps also in the internal ear remains obscure.

Aside from the obstructive features and the ability to harbor infection, attention must be paid to the role of the lymphoid tissue and the lymph channels of the eustachian tube which pass along this tube and end in the retropharyngeal lymph nodes.

CAUSES OF DEAFNESS

Mao and Hughson[2] ascribed the deafness of the 540 children in the Pennsylvania School for the Deaf to the following causes:

Biologic defects	53 per cent
Trauma	14 per cent
Meningitis	17 per cent
Contagious diseases (such as measles, scarlet fever, etc.)	15 per cent
Incidental causes	1 per cent

1. Desjardins, A. U.: Science **75**:569, 1932.

1*a*. Warren, S.: Effects of Radiation on Normal Tissues, Reprint No. 560, Harvard University Cancer Commission, 1942, pp. 42-43.

2. Mao, C. Y., and Hughson, W.: Patency of the Eustachian Tube in Relation to Profound Hearing Loss in Childhood, Arch. Otolaryng. **33**:824-829 (May) 1941.

In regard to the group of 53 per cent whose deafness was ascribed to biologic defects it is felt that the biologic defect may render the individual child's auditory apparatus more susceptible to a variety of disturbances, such as infections of all kinds and degrees. In addition, mechanical factors and nutritional or endocrine imbalances may play a part. To all these the normal person of this age would probably be immune or at least would present a higher degree of resistance so far as the auditory mechanism is concerned. Mao and Hughson stated that in this group of profoundly deaf children neither patency nor obstruction of the eustachian tube is a factor of real importance.

There are, however, other groups of children and adults in whom lymphoid hyperplasia and hypertrophy obstructing the pharyngeal end of the eustachian tube or any portion of the tube will definitely affect the acuity of hearing.

HISTOLOGIC CHANGES DUE TO INFECTION

In chronically inflamed glands there is a tendency toward lymphoid infiltration. In the atrophy resulting from the physiologic decline of some organs, such as the ovary, the prostate gland, the breast and the thyroid gland, and in almost all tissues with atrophy due to chronic inflammation there is infiltration of hypertrophied lymphoid tissue. The function in all likelihood is to remove the debris incident to the slow disintegration; along with this process in any glandular organ there are apt to be focal areas of hypertrophy of mucous glands aimed at compensating for the loss of function of the destroyed glands. Infections play no small part in the pathologic changes.[3] According to J. R. Lindsay,[4] the major cause of deafness in early years is inflammatory disease of the eustachian tube and the middle ear.

These inflammatory diseases appear first as acute infections of the nasopharynx, the nose, the throat and the sinuses, the invading organisms attacking the mucosa and especially the lymphoid tissue.

The inflammatory process in this early stage may be arrested with proper care, provided the patient's vital forces will respond adequately in producing enough immunity or antibodies to counteract the infection. This vital force depends to a certain degree on the function of the thyroid gland. If the infection is not arrested in the early stage, other tissues are invaded, the lymphatic channels are occluded, adequate drainage is blocked, and suppuration occurs. Without relief, the process goes into the chronic state, producing chronic sinus infection and hypertrophy of lymphoid tissue with their sequelae.

3. Konzelman, F., director of the laboratory of the Atlantic City Hospital, Atlantic City, N. J.: Personal communication to the author.

4. Lindsay, J. R.: Eustachian Tube Function and Deafness, Minnesota Med. **62**:250-257 (March) 1943.

APTITUDE TEST

An aptitude test was conducted for the students of the United States Coast Guard Radio School. Some of the findings have a direct bearing on this problem.

We used the audiometer test, clinical examination and a personal history as the basis of our investigation. The graduate students had an average decibel loss of 13.7 for air conduction and 4.4 for bone conduction, while the disenrolees had an average decibel loss of 19.4 for air conduction and 6.3 for bone conduction. Of the graduates, 12.9 per cent had had scarlet fever, while 28.6 per cent of the disenrolees had had this disease; 19.7 per cent of the graduates, compared with 52.0 per cent of the disenrolees, had sinus infection; 9.0 per cent of the graduates, compared with 22.0 per cent of the disenrolees, had epipharyngeal lymphoid hyperplasia.

A mental fatigue test was given all students: The disenrolees' efficiency declined 50 per cent faster than that of the graduates. It was concluded that failures of students to complete the radio school course were due in part to mental fatigue caused by decreased acuity of hearing and by infection.

The aptitude test is superior to the psychologic, intelligence quotient test in rating students in this type of work. Racial characteristics are a definite influencing factor. Unfortunately, we were unable to make basal metabolic rate tests on these students.

INFLUENCE OF THYROID GLAND AND INFECTION

The influence of thyroid gland activity on lymphoid tissue and infection of the upper respiratory tract is marked.

Hyperthyroidism, according to R. C. Moehlig,[5] is frequently associated with hypertrophy of the nasal mucous membranes, polyp formation and allergic states, such as hay fever, urticaria, eczema and asthma. Allergy is related to the thyroid gland through the sympathetic-hypophysial system.

The relationship and influence of the thyroid gland on the pituitary gland is an all-important one, for by this relationship and influence we can understand that the thyroid gland would affect the mesodermal tissue via the pituitary changes. The lymphatic channels, the blood and blood vessels and the reticuloendothelial system are derivatives of the mesoderm.

A. Kocher[5] stated that lymphoid hyperplasia is in direct relation to hyperthyroidism and, according to Boyd, lymphoid tissue shows

5. Kocher, cited by Moehlig, R. C.: Hyperthyroidism, Clinics **1**:1115-1167 (Feb.) 1943.

changes almost as constant in character as those of the thyroid gland itself. Kocher also called attention to the relative lymphocytosis which is found frequently in the peripheral blood, monocytes predominating. The cause of the lymphocytosis has been ascribed to the hyperplasia of the lymphatic system, the enlarged spleen and lymph glands, tonsils and Peyer's patches.

The relationship of hypothyroidism to the lymphoid hyperplasia and infection is never more marked than in hyperactivity of the thyroid gland.

Hypothyroidism occurring in young babies is most difficult to diagnose. However, there are a few leading abnormalities that will help the physician: marked overweight at birth and retardation in teething, walking and talking. If these four signs are present at the same time the diagnosis is almost certain.

In childhood the signs and the symptoms of hypothyroidism become more definite up to the age of 12 years. The patients may have frequent colds and show a poor circulation; the skin is dry, and the hair develops poorly and becomes brittle. The lymphadenoid tissue is quite prominent, as evidenced by enlarged tonsils and postpharyngeal adenoid tissue. If this tissue is removed, in all likelihood it will repeatedly reform, and the younger the child the more apt is this tissue to recur, with more marked hyperplasia, unless scar tissue acts as a limiting factor.

Basal metabolism is almost always found below normal but not as much below normal as in adult patients.

The growth of the child appears to be retarded. Roentgen examination of the various joints will reveal retarded development of the centers of ossification.

Spontaneous adult hypothyroidism is ultimately a chronic disease, and the most important etiologic factors are acute and even chronic infectious diseases. It is more common in women than in men (about 8 to 1). The predisposition of women to have spontaneous hypothyroidism is due to frequent congestion and toxic infectious disturbances of the thyroid gland during sexual life, such as menstruation, pregnancy and menopause.[6]

The effect of infection on the thyroid gland is to alter its metabolism, which in turn reduces the patient's ability to resist further infection.

The signs in a marked case are the swollen face, the thickened lips and nose and the swollen mucous membrances of the nasopharynx. The sebaceous secretions are scant, and cutaneous eruptions of dif-

6. Crotti, A.: Diseases of the Thyroid, Parathyroids and Thymus, Philadelphia, Lea & Febiger, 1938.

ferent kinds are often present. The nervous symptoms are striking: The memory is weakened, the mental processes are slow, and the organs of sense and of reflex are diminished. The voice is more or less husky, owing to edema of the laryngeal mucous membrane. The organs of special sense are quite often affected; hearing, sight, taste and smell are diminished. Deafness to a greater or less degree is common and is due not only to infiltration of the mucous membranes but also, it seems, to disturbances of central origin. Tinnitus is frequent. The temperature is lowered: it varies between 95 and 98 F. The pulse rate varies between 50 and 65, and the respirations are slow. The blood pressure is usually below normal.

In a review of the cases recorded in my office it was found that 19.2 per cent of the patients had thyroid dysfunctions. Lymphoid hyperplasia was present to a marked degree in 9.67 per cent, to a moderate degree in 32.88 per cent and to a slight degree or not at all in 57.35 per cent. Transillumination and roentgen study of the sinuses showed no evidence of infection in 72.2 per cent, moderate clouding in 20 per cent and marked clouding in 7.8 per cent. Many of the patients whose sinuses were not abnormal on such examinations were actually infected with streptococci, pneumococci or other toxin-producing organisms, with a serous discharge. In some cases facultative anaerobic bacteria were found in the sinuses filled with a foul-smelling gas. Females predominated 5 to 1 over males. Hyperactivity of the thyroid gland existed in a ratio of 3 to 1 over hypoactivity. Pulse, temperature, respirations, weight and blood pressure are indicative of the basal metabolic rate, but they are by no means constant. The age is significant in that a slight abnormality of thyroid function produces more marked systemic effects in the child than in the older person. Also, the child responds more quickly and completely to treatment than the older person.

The basal metabolic rate does not always indicate the kind of treatment required to correct the condition. For example: all patients with minus rates do not respond favorably to thyroid therapy, nor do all with plus rates respond favorably to iodine therapy. They may require mixed treatment.

To avoid a mistaken diagnosis it is imperative that a stereotyped routine procedure be followed in examing all new patients. Some of the more frequently neglected procedures will be outlined. The history should include all infectious diseases the patient suffered from and their severity, together with any complicating sequelae, such as arthritis, rheumatism and cardiac and pulmonary dysfunctions. Gastrointestinal disorders and disease of the upper respiratory tract should be noted. Any nervous disturbances should be tabulated and studied.

The routine examination should include determination of temperature, pulse, respiration, blood pressure and reflexes. Sinus infections can almost always be diagnosed after a thorough examination of the nares, transillumination and roentgen study of the sinuses in a case in which there is a history of infection, pain and what is called frequent colds. Lymphoid hyperplasia can be seen in the epipharynx by means of the laryngeal mirror, the nasopharyngoscope, or the illuminating epipharyngoscope which I presented before the American Academy of Ophthalmology and Otolaryngology a few years ago. If any of the cardinal symptoms of thyroid dysfunction are present, a basal metabolic rate test should be performed, also studies of the blood and other tests necessary to determine the function of the thyroid gland.

Patients suffering from hyperplasia of epipharyngeal lymphoid tissue, suppurative sinusitis and dysfunction of the thyroid gland respond readily when the sinus infection is brought under control and the thyroid dysfunction is corrected. With some whose disease is of the more chronic protracted type, with definite hypertrophy of lymphoid tissue and formation of cicatricial tissue, it will be necessary to remove the tissue by surgical procedure, irradiation or diathermy, but not until after the previous treatment has been carried out.

In conclusion, the purpose of this paper is to point out the necessity of diagnosing and correcting thyroid dysfunction and suppurative sinusitis before removing hyperplastic lymphoid tissue by physical means. In the majority of cases the more radical means will be unnecessary.

ACUTE NONSPECIFIC INFECTIONS OF THE PAROTID GLAND

A Review

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DURING the period of the past ten years it has become apparent that the incidence of acute nonspecific infections of the parotid gland has decreased to such a degree that this entity might now be considered within the realm of problems solved.

Mosher[1] has written of the unsolved problems of otolaryngology along with the disappearing diseases. Of the latter he mentioned epidemic sore throat, papilloma of the larynx, fibroma of the vault of the pharynx, tuberculosis of the larynx, and late effects of syphilis manifested by gumma of the soft palate and sinking of the external nose. Perhaps one can justifiably include the acute nonspecific infections of the parotid gland with the disappearing diseases mentioned at the time his article was presented to the Academy of Ophthalmology and Otolaryngology.

One readily recalls the ominous import of this type of disease prior to recent advances in treatment. With added knowledge of the treatment of infections of the parotid gland it has been possible to avoid the frequent end result of death and the incisional damage of skin, gland and facial nerve, save in unusually complicated cases.

The newer methods of diagnosis and treatment brought about by laboratory and biologic advances in all categories of medicine have made one mindful of additional progress in the future. In reading the Scriptures and historical writings one can appreciate the keen observations of men of Hippocrates' time in describing parasites, dropsy and the swollen gland, the parotid gland. It was he who initially described and recorded swelling of the parotid gland, but little did Hippocrates realize the subdivisions of that entity.

A review of acute nonspecific infections of the parotid gland from his time till now would be interesting in relation to the eras of new types of treatment.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc.

1. Mosher, H. P.: Unsolved Problems in Otolaryngology, Tr. Am. Laryng., Rhin. & Otol. Soc. **43**:27, 1937.

It is the purpose of this writing to review all acute nonspecific infections of the parotid gland of the past twenty years seen at St. Luke's Hospital, New York, through the department of otolaryngology. It is not my intention in the review to deal with the various possible causes, whether primary or secondary, but rather to interpret the decrease in the incidence of these infections. The major factors in the decrease are improved water and electrolytic balance, oral hygiene, chemotherapy and biotherapy. The use of penicillin in civilian hospitals was delayed by military necessities, and, too, the lack of patients treated with penicillin has made it impossible to evaluate this means of therapy. One patient was so treated, and the glandular involvement resolved in two weeks without incision. The last case of acute nonspecific infection of the parotid gland occurred late in 1943; hence the discussion of biotherapy will not be included.

Chronologically, the study and subsequent knowledge of water and electrolytic balance come first, inasmuch as they antedate modern hospital oral hygiene and the increased knowledge of the use of chemotherapy. Dehydration has played an important role in conditioning the gland and duct for invading organisms by reason of varying degrees of dryness of the mucous membrane and the inhibition of the peristalsis of Stensen's duct. The dry mucous membrane has been altered in its cellular metabolism and tissue reaction to the presence of microscopic organisms. The correction of depleted body fluids and salts has made it possible to bring about a decrease in the incidence of this type of infection. Water, both intracellular and extracellular, provides the necessary vehicle to make full use of the chemotherapeutic agent. Body fluids and sulfonamide compounds go hand in hand.

The injudicious use of intravenous and parenteral fluids did little good to the dehydrated body with concurrent infection. This subject of water and electrolytic balances will be discussed later in this review.

In recent years hospitals have employed full time dental residents to survey ward and some private patients for oral defects and to remove sources of infection, correct ill-fitting dentures and instruct the patient in the care of the mouth to keep it clean. Inflammatory changes about the opening of Stensen's duct must be prevented, in view of the likely complication of ascending infection. One of the cases reviewed was interesting and bears out this fact of oral hygiene. A note on the chart stated, "Pus from Stensen's duct stopped and the gland subsided following the removal of an ill-fitting upper denture."

Allergy as a cause was considered in this review. Only one case was diagnosed as allergic, though no smear for eosinophils was made. The gland subsided in one week. However, the patient was transferred to another hospital because of toxic encephalitis and arteriosclerotic heart disease. In the section headed "Comment" the subject of chronic

nonspecific recurring parotitis will be mentioned, since the last-mentioned case could possibly fall into this category at a later date.

Coughlin and Gish[2] grouped all acute nonspecific infections of the parotid gland as "acute surgical parotiditis." The various descriptive pathologic terms, as "septic," "gangrenous," "suppurative," "phlegmonous" or "necrotic," are only phases of the disease. In this review the terms "nonsuppurative" and "suppurative" are used only in regard to the chart showing the incidence of cases by years, since the need of incision of glands parallels the incidence of the acute infections of the parotid gland and suggests the virulence of the organisms active that year.

THE ANATOMY

The complex anatomy of the parotid gland makes it difficult to treat these infections surgically without some injury being done to branches of the facial nerve. The facial nerve enters the gland after making its exit from the styloid foramen. It runs through the gland substance until it forms the plexus parotideus and the distal branches that fan out to form the temporal, zygomatic, buccal, mandibular and cervical branch endings. This network of facial nerve twigs in close relationship with the gland proper makes it difficult to secure adequate drainage by incision without severing some of the nerve fibers with resultant motor loss to the area served by those fibers.

The gland is composed of the individual lobes, certain portions of the external carotid artery, the superficial part of the facial nerve, and their branches. The gland is covered by a very hard capsule that is continuous with the deep cervical fascia. Its weakest point is the medial-superior surface in close contact with the buccal mucosa. This is a choice region for evacuation of a localized infection of the parotid gland by reason of it being possible to secure drainage without incision of the skin and possible damage of the nerve. Of course, the deep blood vessels must be avoided in this approach.

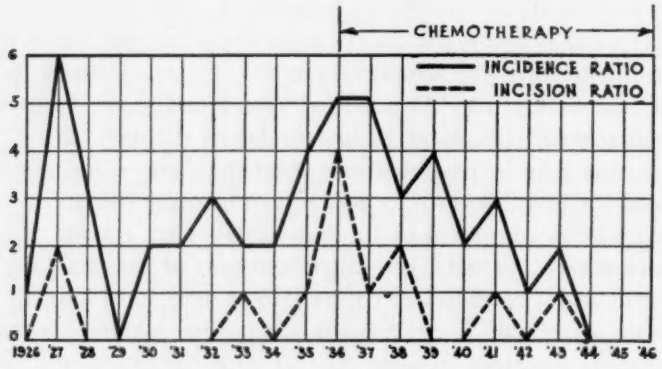
Stensen's duct is composed of a heavy fibrous coat and is lined with short columnar cell epithelium. Between these layers lie contractile fibers that give rise to peristaltic motions of the duct, which are innervated from the plexus sympatheticus on the carotid artery, the facial nerve, the auriculotemporal nerve and the great auricular nerve. Gray stated that while the others are branches of the facial nerve, probably the branch from the auriculotemporal nerve is derived from the glossopharyngeal nerve via the otic ganglion. The duct crosses the masseter muscle and then is combined with its smaller ducts, pierces the buccinator muscle and emerges through the mucosa

2. Coughlin, W. T., and Gish, E. R.: Acute Surgical Parotitis, Arch. Surg. 45:361 (Sept.) 1942.

of the oral cavity to form the opening of the ductus parotideus. It derives its blood supply from the external carotid artery and its local branches.

TWENTY YEAR REVIEW

This review is inclusive of all acute nonspecific infections of the parotid gland from January 1926 to October 1946. The patients admitted to all services during this period totaled 175,653. The number admitted monthly during this period was fairly consistent and never was more than 400, save the four individual years that did not influence the incidence-admission ratio materially. The number of acute nonspecific infections of the parotid gland totaled 50. As an interesting comparison, 32 specific infections of the parotid gland were recorded, these occurring accidentally after admission of the patients to the hospital, mostly on the pediatric service. The sex incidence was 26



The decreased incidence of acute infections of parotid glands.

for males and 24 for females, and of these 5 were children. The glandular involvement was single in 39 cases and double in 11.

The bacteriologic study revealed the following organisms: nonhemolytic streptococci, Staphylococcus aureus, pneumococci, Bacillus coli, gram-positive cocci, gram-positive diplococci and one acute granuloma of syphilitic origin. The relative numbers of the different organisms are of no great value statistically.

Of the group, 4 infections were postoperative, 1 following iridectomy, 1 following operation for diverticula of the duodenum, and 2 resulting from cholecystectomy. This is mentioned only for interest, as all acute nonspecific infections of the parotid gland are considered as one group, the incidence, the treatment and the diagnosis not depending on previous surgical procedures. This method of considering infections of the parotid was suggested by Coughlin and Gish.[2]

In this group 7 were considered as unresolved by reason of the facts that death was due to pneumonia, arteriosclerotic heart disease, carcinoma of the colon with metastasis and carcinoma of the prostate with pulmonary metastasis, and 2 patients left the hospital early in the course of the acute glandular involvement. One of these patients gave a history of an acute glandular involvement one year prior to admission lasting about a month.

The chart shows the yearly incidence of acute nonspecific infections of the parotid gland. It is interesting to note that the need of incision is parallel to the rate of incidence. The reason for the apparent low incidence during the years prior to 1935 may be due to the gradual change over to the unit system in recording hospital and clinic patients. This system of cross filing has made for more comprehensive inclusion of all diagnoses. The decrease in incidence since 1935 has been gradual, and there have been no new cases of acute nonspecific infection of the parotid gland since late in 1943.

COMMENT

The decreased incidence of acute nonspecific infections of the parotid gland has been made possible by the increased knowledge of water and electrolytic balance, the correction of oral sources of infection and the unfolding use of chemotherapeutic agents. To a certain extent these improved methods of treatment have progressed in that order, and in the discussion they will be covered in that sequence.

In 1936, at the University of Michigan, Coller and Maddock[3] published the results of their work after an exhaustive study of water and electrolytic balance. From that study it was learned that water need is definitely related to body weight; i.e., 65 per cent of body weight is made up of water, and any increase or decrease of that amount is evidence of disturbance of water balance. This may be determined by laboratory methods. In a general way, one may roughly estimate the intake-output balance in the normal man. However, in febrile or metabolic disease the need for an above normal intake of water necessitates a more thorough estimate of the water needs.

At a normal metabolic rate the output is about 250 cc. from the kidneys, 200 cc. from stools and 1,500 cc. by vaporization, a total of 2,000 cc. With the output of 2,000 cc., the intake should equal or surpass that amount. The minimum intake for the kidney to carry on its function is 500 cc. a day. A scanty output of high specific gravity is indicative of decreased water metabolism. The more normal the intake, the more likely that the kidneys can carry on their excretory

3. Coller, F. A., and Maddock, W. G.: Water and Electrolyte Balance, Surg., Gynec. & Obst. **70**:340 (Feb.) 1940.

processes. This is important when chemotherapy is being used, for the kidneys must be enabled properly to excrete the sulfonamide crystals. The febrile patient or the one with hyperthyroidism uses about 3,000 cc. of fluid a day. Operative procedures on a hot day will cause the patient to lose at least 3,000 cc. of water on that day.

The principal intake of water is that ingested plus the water derived from oxidation products. This intake goes into three compartments, the blood, the intracellular and the extracellular space.

Besides the establishment of normal water balance, the electrolytic balance must also be corrected if there should be a diminution of the blood chlorides from the normal of 570 to 620 mg. per hundred cubic centimeters. The determination of the acid-base equilibrium by means of carbon dioxide—combining power is important, to estimate the need of supplementing the chlorides in order to avoid acidosis. The daily estimate of the sodium chloride utilized in metabolism is 11 grains (0.7 Gm.) a day. Experimentally it has shown that early shock may be obtained by infusion of more than this amount.

When there has been loss of body proteins in the forms of albumin and globulin, it is necessary to make studies of blood plasma to rule on the possibility of hyperproteinemia or hypoproteinemia. The function of blood plasma protein is to maintain normal osmotic pressure in the capillaries. Normally the osmotic pressure of the blood plasma is such that the fluids tend to leave the tissues and enter the blood, but this is counteracted by the capillary blood pressure. A decrease in blood plasma will mean less tissue fluids entering the blood stream and consequent storing of fluids in the tissues, causing edema.

In recent years the full time dental residents added to hospital staffs have done much to abolish sources of infection, to correct ill-fitting dentures and to educate the patient to maintain oral hygiene religiously. Mucous membranes of the mouth which have altered cellular metabolism because of dryness are prone to become infected by organisms present in the mouth.

Chemotherapy has been of considerable value in the treatment of acute nonspecific infections of the parotid gland. Before this new means of therapy, treatment of a conservative type consisted of external hot and cold applications, massage of the gland, dilation and suction of Stensen's duct, injecting of antiseptic solutions into the duct and the use of chewing gum to excite secretion of fluids in the gland. However, in acute inflammatory conditions chemotherapy brought about a more rapid subsidence of the glandular infections, which in turn obviated the need for incision of the gland. The administering of sulfonamide compounds to a patient with normal water and electrolytic balance at the first sign of parotid swelling has not

only decreased the degree of inflammation but shortened the duration of the pathologic involvement. The early use of chemotherapy decreases the likelihood of pus formation.

In this review it has been shown that these cases were instances of acute nonspecific infections of the parotid gland. However, there is no guarantee that in the future some of them may not become instances of chronic recurring nonspecific parotitis. McCaskey[4] has mentioned this type of entity with the possible causes, i.e., lodging of foreign bodies in the duct, hyperplasia of the gland tissue occurring with diseases of the genital tract and of some of the endocrine glands, salivary retention in newborn infants, malaria, drug intoxication, specific infections such as tuberculosis, actinomycosis and syphilis, and allergy. These are possibilities, though in his conclusion he arrived at no definite opinion as to an etiologic factor. Biopsy demonstrated the chronic recurrence of pathologic involvement of the parotid gland. Fowlkes, discussing the paper of McCaskey,[4] made note of the frequency with which mental instability and mild psychosis were found in patients with chronic recurring nonspecific infections of the parotid gland seen by him at Bellevue Hospital. Earlier in this paper mention was made of a patient who had an acute infection of this gland that subsided but who was transferred to another hospital because of toxic encephalitis. It may well be that the glandular infection of this patient will eventually be found in the category of the latter type of nonspecific infections of the parotid gland.

CONCLUSIONS

1. There has been a decrease in the incidence of acute nonspecific infections of the parotid gland.

2. Improved use of water and electrolytic agents has corrected varying degrees of dehydration in patients with such infections.

3. Proper oral hygiene, the correction of ill-fitting dentures and the removal of sources of infection have protected the parotid duct from being invaded by organisms.

4. Chemotherapy and the improved water balance provided with it have greatly decreased the degree of pathologic involvement of the gland and shortened the duration of the infection.

4. McCaskey, C. H.: Chronic Non-Specific Recurring Parotitis, Tr. Am. Laryng., Rhin. & Otol. Soc. **48**:56, 1942.

BARRIER MEMBRANE OF THE COCHLEAR AQUEDUCT

Histologic Studies on the Patency of the Cochlear Aqueduct

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IT IS generally assumed that the cochlear aqueduct is a canal through which spinal fluid flows from the subarachnoid spaces to the scala tympani of the cochlea. This theory of the physiology of the cochlear aqueduct is based entirely on histopathologic observations. Grunberg, Voss, Ulrich, Grove, Crowe, and Perlman and Lindsay[1], among others, collected a number of temporal bones of patients who succumbed soon after an intracranial operation or a fracture of the skull. When blood was found in the subarachnoid spaces of these specimens, blood was also noted in the perilymphatic spaces of the cochlea. In addition, red blood cells were demonstrated in the cochlear aqueduct, especially near its cochlear opening. From these observations Crowe deduced that "there is a normal flow of cerebrospinal fluid in the cochlear aqueduct from the meninges to the scala tympani."

Experiments done on animals and cadavers up to the present time have led to much confusion about the patency of the cochlear aqueduct, due to the numerous artefacts invariably present in these studies.[2]

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1. (*a*) Grunberg, K.: Zur Frage der Existenz eines offenen Ductus perilymphaticus, Ztschr. f. Hals-, Nasen- u. Ohrenh. **2**:146, 1922. (*b*) Voss, O.: Klinische und pathologisch-anatomische Folgeerscheinungen geburstraumatischer Schädigungen des Felsenbeins, Monatschr. f. Kinderh. **34**:568, 1926. (*c*) Ulrich, K.: Verletzungen des Gehörorgans bei Schadelbasisfrakturen, Acta oto-laryng., 1926, supp. 6, p. 1. (*d*) Grove, W. E.: Otologic Observations in Trauma of the Head. Arch. Otolaryng. **8**:249 (Sept.) 1928. (*e*) Crowe, S. J.: Pathologic Changes in Meningitis of the Internal Ear, ibid. **11**:537 (May) 1930. (*f*) Perlman, H. B., and Lindsay, J. R.: Relation of the Internal Ear Spaces to the Meninges, ibid. **29**:12 (Jan.) 1939.

2. Weber-Liel: Experimenteller Nachweis einer freien Communication der endolymphatischen und perilymphatischen Raume des menschlichen Ohrlabyrinthes mit extralabyrinthischen intracraniellen Raumen, Virchows Arch. f. path. Anat. **77**:207, 1879. Retzius, M. G.: Das Gehörorgan der Wirbelthiere, Stockholm,

During systematic studies of human and animal temporal bones, especially those with subarachnoid hemorrhages, I made observations which seem to contradict the foregoing conclusions as to the patency of the cochlear aqueduct.

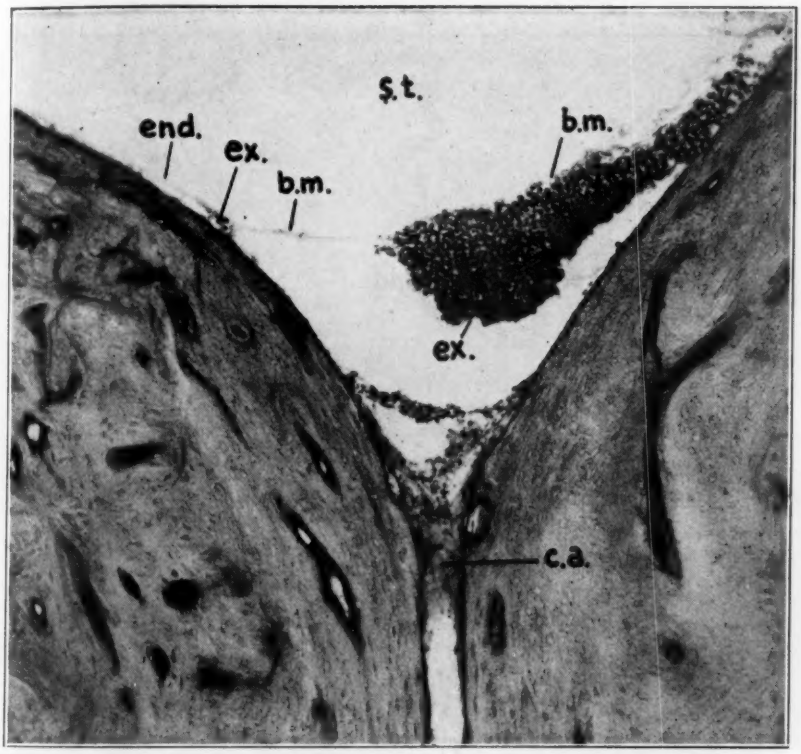


Fig. 1 (case 1).—The patient died of suppurative leptomeningitis. Exudate (*ex.*) penetrates through the cochlear aqueduct (*c.a.*) and pushes the thin barrier membrane (*b.m.*) into the lumen of the scala tympani of the cochlea (*s.t.*). Note the infiltrating pus cells under the neighboring endosteum (*end.*).

OBSERVATIONS

Case 1.—A 53 year old man died of fulminating suppurative leptomeningitis which apparently followed an ordinary cold. The spinal fluid pressure was 400 mm. of water a few hours before death. Autopsy was performed one and a half hours post mortem.

Vertical serial sections showed the cochlear aqueduct to be filled with purulent exudate up to its cochlear opening. No exudate, however, was found in the peri

Samson & Wallin, 1884. Siebenmann, F.: Die Korrosionsanatomie des knochernen Labyrinthes des menschlichen Ohres, Wiesbaden, J. F. Bergman, 1890. Karlefors, J.: Der Aquaeductus cochleae beim Menschen, Acta oto-laryng., 1924, supp. 4, p. 145. Meurman, Y.: Zur Anatomie und Physiologie des Aquaeductus cochleae, Acta Soc. med. fenn. duodecim, s.B. **13**:175, 1931.

lymphatic spaces of the cochlea. All the exudate was accumulated under a thin membrane measuring less than 1 micron in thickness. This membrane seemed to separate the cochlear opening of the aqueduct from the lumen of the scala tympani. The exudate pushed the membrane toward the lumen of the scala tympani and also infiltrated between the neighboring endosteum of the scala tympani and the endosteal bone (fig. 1).

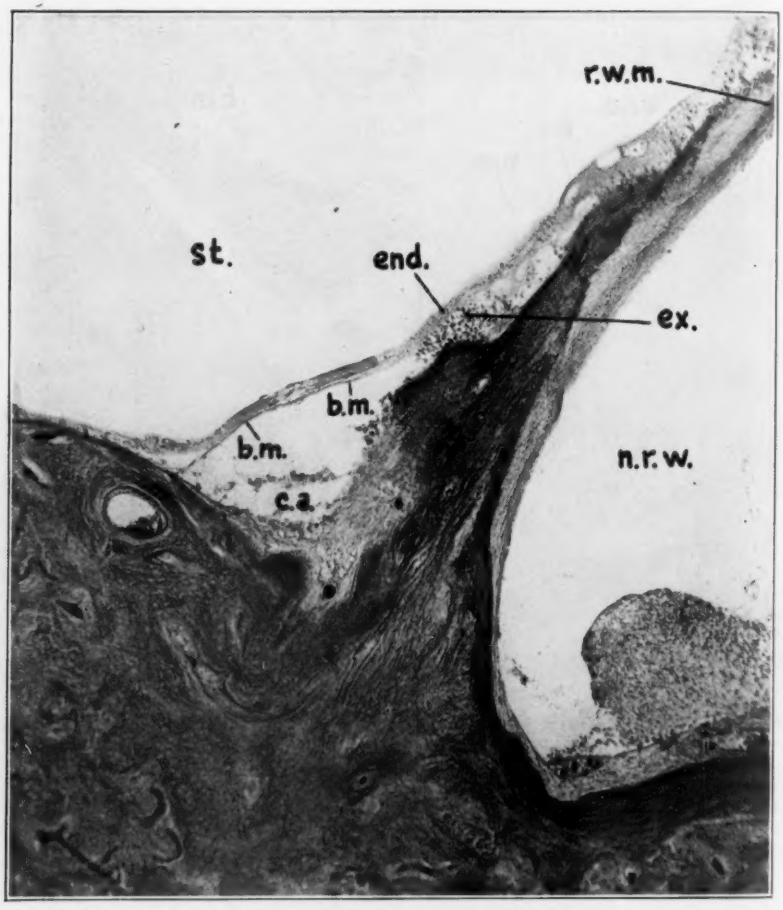


Fig. 2 (case 2).—The patient died of purulent leptomeningitis and subarachnoid hemorrhage. Exudate (*ex.*) passing through the cochlear aqueduct (*c.a.*) is accumulated under the barrier membrane (*b.m.*) and between the endosteum (*end.*) and the bony wall of the scala tympani (*s.t.*). *r.w.m.* denotes the round window membrane; *n.r.w.*, the niche of the round window.

Case 2.—A 62 year old woman died of purulent leptomeningitis following acute Pneumococcus type III infection of the middle ear. The spinal fluid pressure was 600 mm. of water a few hours before death. Autopsy, performed four hours after death, revealed subarachnoid hemorrhage in addition to purulent leptomeningitis.

Horizontal serial sections of the right temporal bone showed purulent exudate and blood in the cochlear aqueduct, extending to its cochlear opening, where the

exudate and the blood were stopped by a membrane separating the aqueduct from the basal turn of the scala tympani. Blood and exudate could be traced from the aqueduct to the midportion of the round window membrane, at which place they were located between the swollen endosteum and the lamina propria of the round window membrane. From this area the exudate extended along the under surface of the basilar membrane, in which location a distinct endosteum was not recognizable (fig. 2).

CASE 3.—A 48 year old woman was admitted to the Presbyterian Hospital in coma and died shortly after admission. Autopsy, performed seven hours post mortem, revealed a cyst of the third ventricle of the brain.

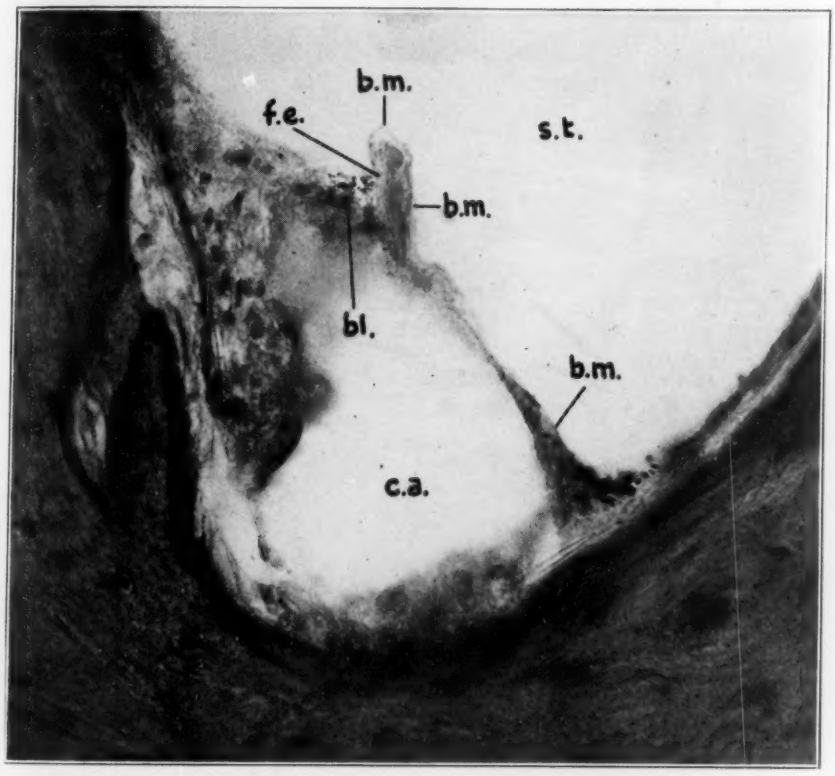


Fig. 3 (case 3).—A barrier membrane (*b.m.*) separates the opening of the cochlear aqueduct (*c.a.*) from the scala tympani of the cochlea (*s.t.*) in a 48 year old woman. Blood (*bl.*) has accumulated under the ruptured and folded end (*f.e.*) of the barrier membrane.

In horizontal serial sections of the left temporal bone, blood was found in the wide, inferior portion of the cochlear aqueduct. A pool of blood was seen under a separating membrane at the cochlear opening of the aqueduct. No blood was found in the scala tympani. The membrane was ruptured at one end near its continuation into the endosteum and was folded on itself. Most of the blood cells accumulated right under the ruptured part of the separating membrane. Measurements of the floating folded portion of the membrane indicated that, if straightened out to its apparent former position, it would just reach the point of its previous attachment to the endosteum of the bony wall of the scala tympani (fig. 3).

Two temporal bones obtained in cases 4 and 5 are representative specimens in our collection showing the presence of a continuous membrane which separates the cochlear aqueduct from the scala tympani.

CASE 4.—A young man, 22 years old, died of obstruction of the small intestine. Autopsy was performed four and a half hours post mortem.

In horizontal serial sections a membrane was seen overbridging the cochlear opening of the cochlear aqueduct and separating it from the lumen of the scala

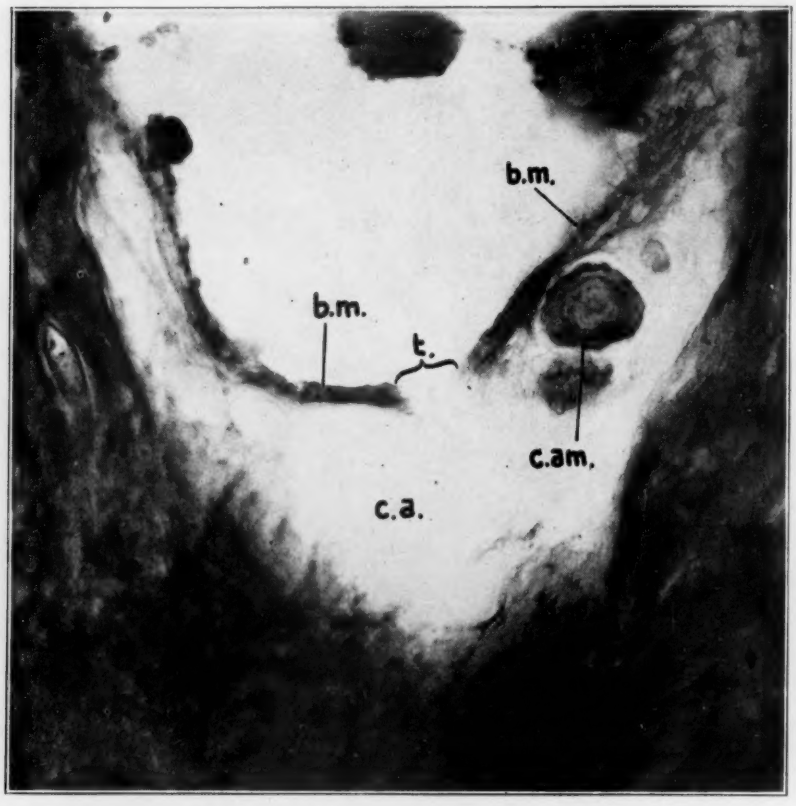


Fig. 4 (case 4).—The cochlear opening of the aqueduct (*c.a.*) contains a barrier membrane (*b.m.*), under which a fully developed corpus amylaceum (*c.am.*) is visible. There is a small tear in the the middle of of the membrane (*t.*).

tympani. This membrane was directly continuous with the endosteum covering the bony walls of the scala tympani. The region of the cochlear opening of the aqueduct was studied in all consecutive serial sections (fig. 4).

CASE 5.—A woman 57 years old died of carcinoma of the liver. Autopsy was performed one hour post mortem.

Vertical serial sections of the right temporal bone showed a very thin membrane separating the cochlear aqueduct from the perilymphatic spaces of the cochlea. This membrane consisted of one to two parallel layers of cells containing elongated, flat nuclei. The membrane was found to be ruptured near its attach-

ment to the bony wall at one side but both ends were directly continuous with the endosteum of the scala tympani (fig. 5).

CASE 6.—A 56 year old woman gave a history of Paget's disease of the skull. She also gave a history of deafness of five years' duration, the loss of hearing becoming more acute during the last month of her life. She died of lobar pneumonia and edema of the lung.

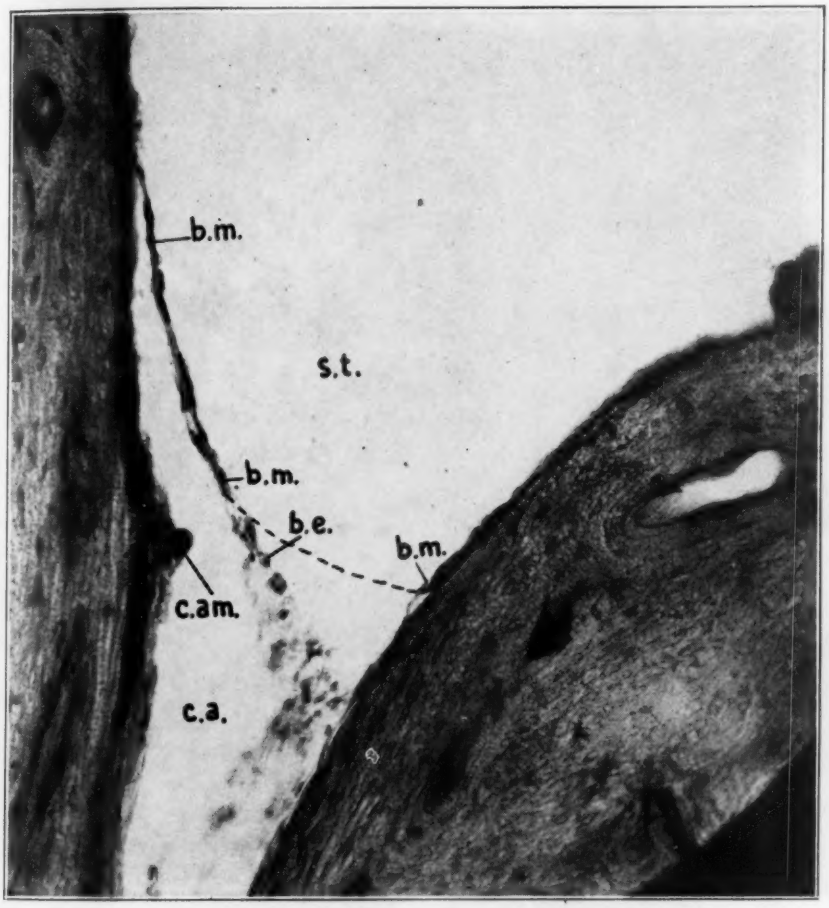
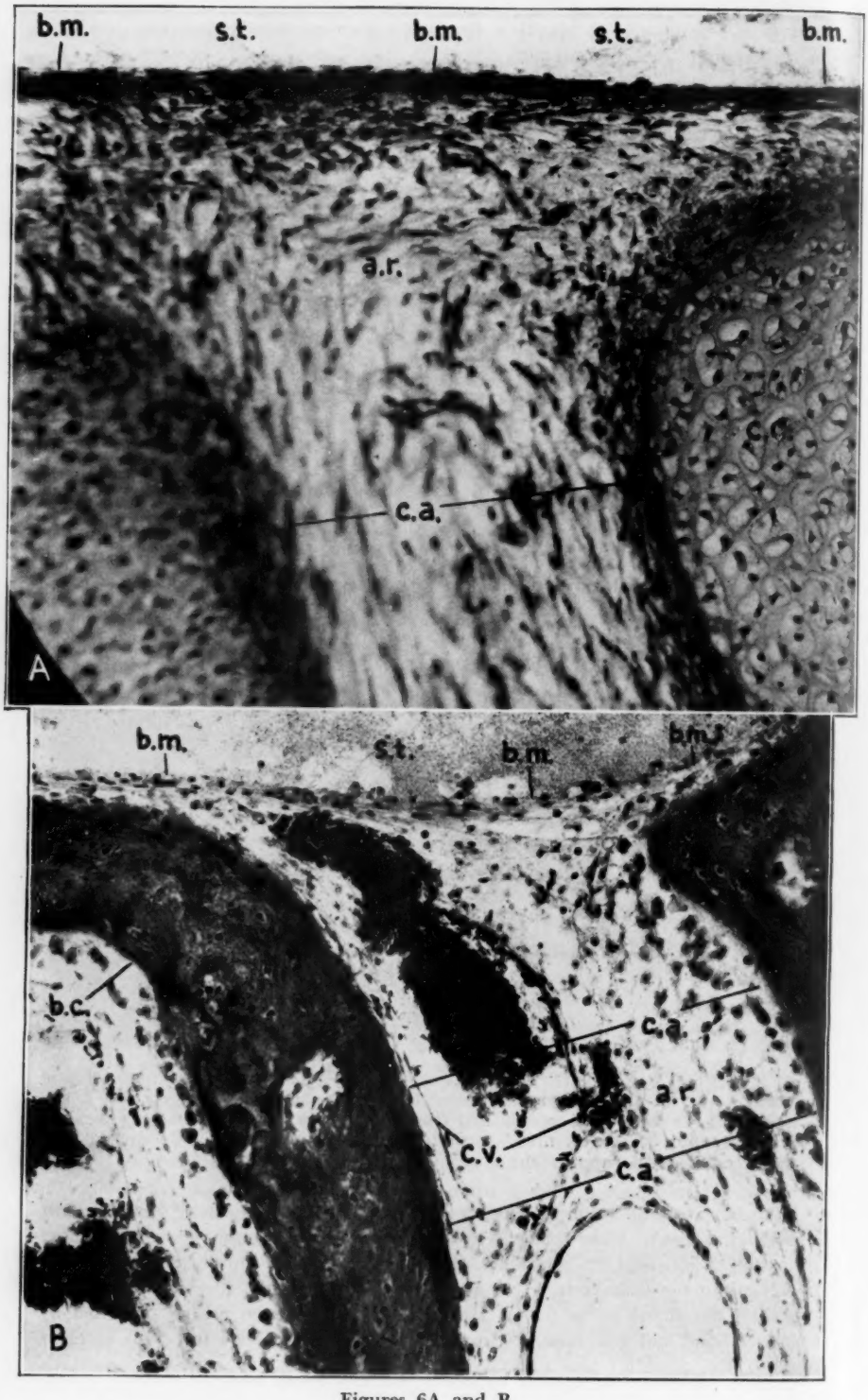


Fig. 5 (case 5).—A barrier membrane (*b.m.*) separates the opening of the cochlear aqueduct (*c.a.*) from the lumen of the scala tympani of the cochlea (*s.t.*). On one end the membrane is detached from the bony wall. The letters *b.e.* denote the broken end of the membrane; *c.am.*, a corpus amylaceum. The dotted line shows the original position of the membrane.

Histologic examination of the left temporal bone in horizontal serial sections showed that newly formed Paget's bone had invaded and completely obliterated the cochlear aqueduct for a distance of 0.6 mm., starting 1.6 mm. from its cochlear opening into the scala tympani. Immediately below this level the bony wall of the aqueduct was found to be partially destroyed and its lumen greatly narrowed by invading bone and granulation tissue. From the 1.6 mm. level up to its cochlear opening the aqueduct was normal and contained typical arachnoidal reticulum.



Figures 6A and B
(See legend on opposite page)

Considerable hemorrhage was visible in the cuplike opening of the aqueduct into the scala tympani. In the cochlear duct of the basal turn there was a small hemorrhage lining the lateral wall, starting at the stria vascularis. No blood was found in the cochlear aqueduct in any of the sections below the level of complete obliteration, although both the dura periosteum and the arachnoidal reticulum were well preserved.

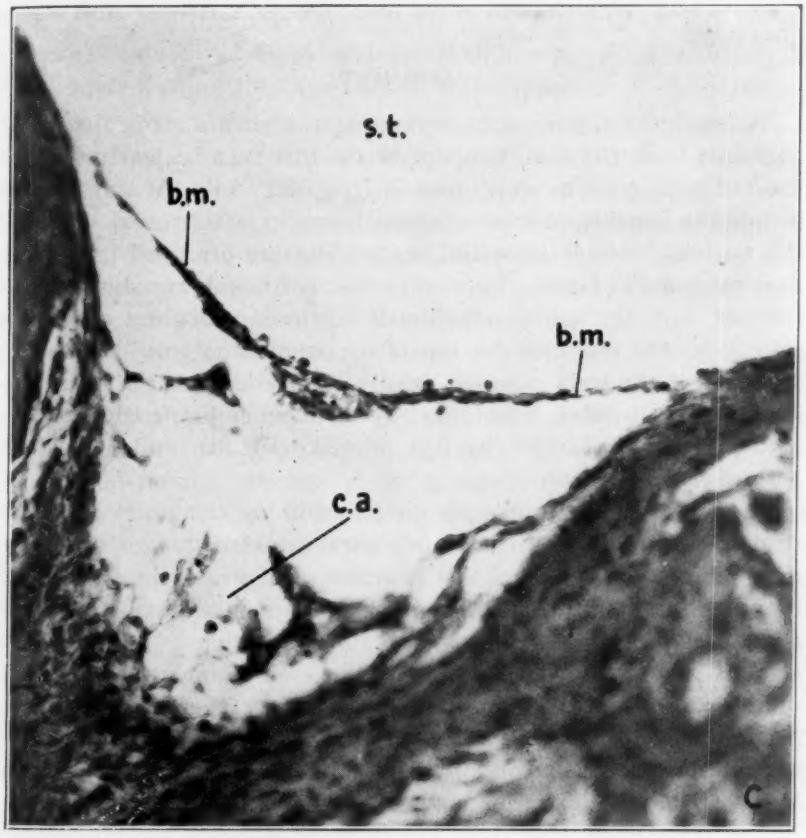


Fig. 6.—*A* shows a barrier membrane (*b.m.*) separating the cochlear aqueduct (*c.a.*) from the scala tympani of the cochlea (*s.t*) in a 110 mm. (crown-rump) human fetus. The membrane is made of two to three parallel layers of elongated cells. The letters *c.c.* designate the cartilaginous capsule of the cochlea; *a.r.*, the arachnoidal reticulum of the cochlear aqueduct.

B shows the cochlear aqueduct (*c.a.*) of a 180 mm. (crown-rump) human fetus, at the cochlear aperture of which a separating membrane (*b.m.*) is visible. Note the parallel orientation of the cells of the membrane. The letters *s.t.* designate the scala tympani of the cochlea; *b.c.*, the bony capsule of the labyrinth; *c.v.* the cochlear vein; *a.r.*, the arachnoidal reticulum of the aqueduct.

C shows a membrane (*b.m.*) separating the aperture of the cochlear aqueduct (*c.a.*) from the scala tympani of the cochlea (*s.t.*) in a 200 mm. (crown-rump) human fetus. The membrane consists of one to two layers of cells containing fusiform nuclei.

The bulb of the jugular vein was unusually large, measuring 8 by 8 mm. in some sections, and extended upward to the level of the posterior semicircular canal. It occupied so much space in the petrous bone that its wall came in direct contact with the middle portion of the cochlear aqueduct.

Examination of the right temporal bone revealed that newly formed bone replaced a large section of the cochlear aqueduct, completely obliterating the narrow portion. A moderate number of red blood cells were seen in the wide, inferior portion below the level of the obliteration. There was no blood in the cochlea.

No pathologic displacement of the round window membrane was noticed on either side.

COMMENT

A continuous membrane separating the cochlear opening of the aqueduct from the scala tympani of the first turn is clearly visible in the 110 mm. (crown-rump) human fetus (fig. 6*A*). At this stage the membrane consists of two to three layers of arachnoidal type cells, the nuclei of which are ovoid shaped and are arranged horizontally in dense parellel layers. This well organized membrane shows a sharp contrast with the underlying loosely scattered reticulum cells of the aqueduct. On one end the separating membrane goes directly over the wall of the scala tympani on the other end, into the inner layer of the round window membrane. A similar membrane is equally evident in the 130, the 180 (fig. 6*B*), the 200 (fig. 6*C*) and the 300 mm. (crown-rump) human fetus.

A similar continuous separating membrane can often be demonstrated (figs. 1 to 5) in adult temporal bones, although no description and demonstration of this structure or of its development can be found in the literature. The name "barrier membrane of the cochlear aqueduct" (membrana limitans aperturae internae aqueductus cochleae) is proposed for this structure. The endosteum covering the wall of the scala tympani at the edge of the inner aperture of the cochlear aqueduct measures 1 micron or less in thickness and consists of a single layer of cells containing very flat nuclei. The barrier membrane of the cochlear aqueduct is a direct continuation of the neighboring endosteum (figs. 1 to 5). In some instances the reticulum of the aqueduct is in contact with the barrier membrane, which can be recognized only by the parallel orientation of its cell nuclei. In other temporal bones the barrier membrane is clearly separated from the underlying reticulum by an empty space. In early childhood the membrane in general seems to be thicker than in adult life.

In many cases all consecutive sections of the region have to be studied in order to demonstrate this membrane. Early postmortem fixation and good decalcification of the temporal bone are important factors. Tearing of Reissner's membrane is frequent during histologic preparation. It is not surprising that tearing of the barrier membrane is even more frequent, because it is thinner and covers a much smaller

area. In rabbits a continuous membrane separating the opening of the cochlear aqueduct from the scala tympani is a standard histologic feature. Immediate postmortem fixation and easier decalcification of rabbit temporal bones probably account for this fact. The extent to which the described membrane closes the opening of the cochlear aqueduct to fluids can be made certain by reconstruction studies only. From the evidence gained from observations in cases 1 to 6 it seems quite probable that the separating membrane forms a complete barrier between spinal fluid and perilymph and prevents the spinal fluid from flowing into the cochlea under physiologic circumstances.

Rapid increase of the pressure in the subarachnoid spaces may result in pathologic fissures in the dense reticulum of the cochlear aqueduct and in rupture of the barrier membrane at the cochlear opening of the aqueduct. The role of the dense reticulum located near the barrier membrane and of the corpora amylacea of the aqueduct seems to be to slow down the flow of spinal fluid in order to protect the delicate barrier membrane against sudden increase of pressure. Bagley's[3] studies on dogs showed that sterile meningitis developed after blood had been injected into the cisterna magna. Similarly a highly increased pressure of spinal fluid will follow subarachnoid hemorrhages which were originated by cranial trauma or neurosurgical operation. Rapidly increasing pressure of the spinal fluid may rupture the barrier membrane of the aqueduct, which can yield to pressure only to a limited degree, because of its shortness. Thus, a pathologic communication may be opened between spinal fluid and perilymph, and blood may be carried by the spinal fluid into the scala tympani.

The foregoing explanation is supported by certain histopathologic developments which are occasionally observed in temporal bones following subarachnoid hemorrhages or purulent meningitis. In these instances, the barrier membrane which separates the lumen of the cochlear aqueduct from the perilymphatic spaces of the cochlea is lifted away from the opening of the aqueduct, and sometimes the neighboring endosteum of the scala tympani shows similar displacement. The blood or the exudate, after having passed through the cochlear aqueduct, is accumulated under the separating membrane. Cases, 1, 2 and 3 demonstrate the pathologic displacement of the barrier membrane following rapid increase of spinal fluid pressure, which was 400 mm. in case 1 and 600 mm. in case 2.

If the pressure of spinal fluid is not high enough to rupture the barrier membrane of the cochlear aqueduct, blood cannot enter the cochlea by this route. This explains the observations made by Perl-

3. Bagley, C.: The Grouping and Treatment of Acute Cerebral Traumas, Arch. Surg. **18**:1078 (April) 1929.

man and Lindsay,[1f] who found unimpaired hearing in a number of patients who had undergone craniotomy.

Four facts support the intravital origin of the pathologic displacement of the barrier membrane and the endosteum: 1. Blood and exudate were found under the barrier membrane and also between the neighboring endosteum and the bony wall of the scala tympani but not in the lumen of the scala tympani (cases 1 and 2). 2. Blood and exudate lined the round window membrane and the under surface of the basilar membrane in case 2, but no blood cells were found in the center of the scala tympani. Only under intravital pressure could blood and exudate force their way under the endosteum of the bony wall and the round window membrane along such an extensive circuitous route. 3. The dense reticulum of the cochlear aqueduct was found to be well preserved in these specimens up to the cochlear opening of the aqueduct. This again testifies in favor of the pathologic nature of the displacement of the barrier membrane. 4. In case 3 the accumulation of blood located just under the ruptured portion of the separating membrane points to a causative relationship between increased spinal fluid pressure and rupture of the barrier membrane.

Many times blood can be traced through the fundus of the internal auditory meatus and the modiolus into the perilymphatic spaces of the cochlea after subarachnoid hemorrhages.[4] Oddly enough, previous workers did not claim that physiologically the spinal fluid flows along the nerve and vessel canals of the internal auditory meatus into the cochlea. If the fact that blood has passed through the cochlear aqueduct is accepted as proof of the latter's patency, so the fact that blood has passed through the modiolus should prove that spinal fluid passes via nerve and vessel canals.

In case 6 the cochlear aqueduct of the left temporal bone was completely obliterated in its narrow portion, measuring 0.6 mm. in length by newly formed Paget bone. In order to avoid technical errors, all the consecutive serial sections, measuring 16 to 22 microns in thickness, were studied. In spite of the complete obstruction of the aqueduct, blood was found in the cochlear opening of this structure. In this temporal bone the absence of blood in the lower, wide portion of the aqueduct, below the level of the obstruction, is additional proof that the blood inside the scala tympani of the cochlea did not pass through the aqueduct. Therefore, the fact that blood has been seen in the "typical" location at the cochlear opening of the aqueduct should not invariably be considered as evidence that it passed through the aqueduct from the subarachnoid spaces.

Complete bony obliteration of the narrow portion of the aqueduct was found on the right side in case 6. There was, however, no evi-

4. Animal experiments designed to clarify this question have been undertaken.

dence of blood in the scala tympani. The fact that blood was present in the wide portion of the right cochlear aqueduct below the level of the obliteration is noteworthy. It indicates that there is a regular flow of spinal fluid which at times may carry blood into the cochlear aqueduct. It does not necessarily mean that spinal fluid reaches the perilymphatic spaces under physiologic circumstances. In this particular instance the cochlear aqueduct was obliterated in both temporal bones,

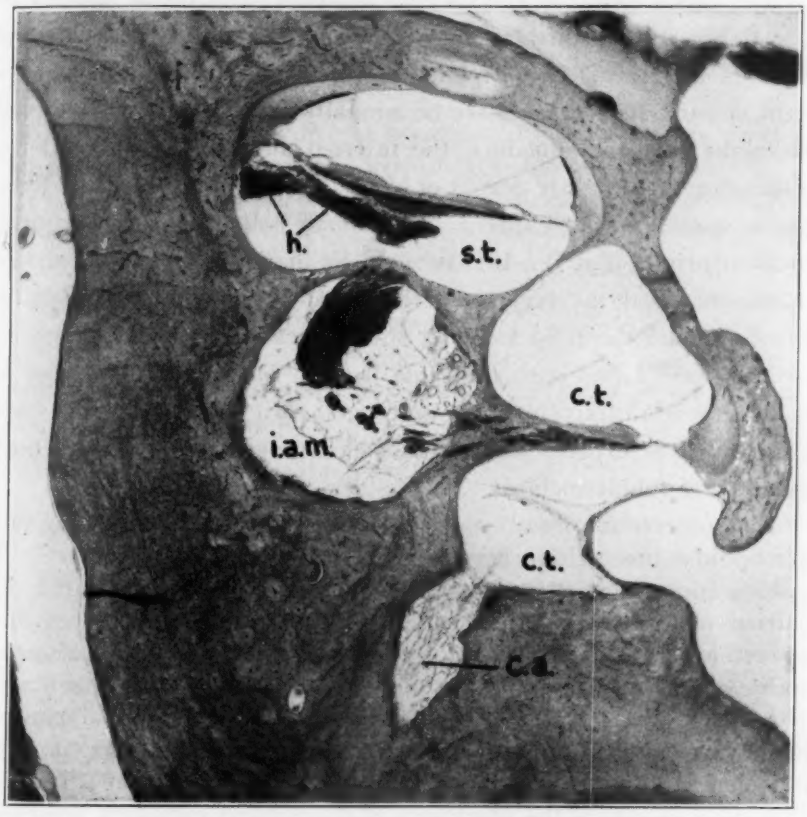


Fig. 7.—Hemorrhage (*h.*) may be seen in the scala tympani (*s.t.*) of a rabbit killed with chloroform. Note the absence of blood in the cochlear aqueduct (*c.a.*), in the basal and second cochlear turn (*c.t.*) and in the internal auditory meatus (*i.a.m.*)

but blood was present in the scala tympani of the left side only, where an intracochlear hemorrhage originating from the spiral ligament was evident. This case supplies additional evidence almost with the clarity of a well planned experiment against the free flowing of spinal fluid through the cochlear aqueduct.

The round window membrane did not show any pathologic displacement in case 6. Were there a normal flowing of spinal fluid

through the cochlear aqueduct into the perilymphatic spaces of the cochlea, one would expect some change in the position of the round window membrane. This membrane represents, in addition to the short and firm ligament of the stapes, the only soft part of the perilymphatic spaces capable of yielding to increased or decreased perilymphatic pressure.

The blood found in the cochlear aqueduct and in the cochlea in case 6 was most probably of agonal origin. There is experimental evidence supporting this explanation. In rabbits killed with chloroform, massive hemorrhages are occasionally seen in the scala tympani while the cochlear aqueduct, the internal auditory meatus and the subarachnoid spaces are devoid of blood. Blood found in the perilymphatic spaces of these experimental animals is beyond doubt of intracochlear origin (fig. 7). It is noteworthy that both in rabbits and in human material the regions of the spiral ligament and the basilar membrane are the most frequent sites of hemorrhages. Although all animals were killed with the same technic, only a small number of them showed intracochlear hemorrhages. Microscopic studies, however, failed to explain why agonal hemorrhages are so rare in both human and rabbit cochleas.

The observation that blood is present in the cochlea following subarachnoidal hemorrhage may be explained in one of two ways: 1. A sudden increase of the spinal fluid pressure may have ruptured the barrier membrane at the cochlear entrance of the aqueduct and opened a pathologic pathway for the spinal fluid. 2. An agonal intracochlear hemorrhage may have occurred. Therefore, the presence of blood in the scala tympani of the cochlea associated with subarachnoid hemorrhage should not be accepted as proof of a physiologic flowing of spinal fluid into the perilymphatic spaces via the cochlear aqueduct. Previous concepts of the physiology of the cochlear aqueduct, however, were based on evidence of such pathologic specimens.

Under physiologic conditions a diffusion, if any, between spinal fluid and perilymph is more likely to take place through the barrier membrane than by simple flowing of spinal fluid into the scala tympani. The separating membrane described seems to be a normal histologic feature of the cochlear aqueduct in a great number of cases and probably represents a barrier between spinal fluid and perilymph.

Additional experimental studies free of artefacts are indispensable in order to clarify fully the physiology of the cochlear aqueduct.

SUMMARY

A membrane separating the lumen of the cochlear aqueduct from the perilymphatic space of the cochlea is demonstrated in human fetuses for the first time.

This separating membrane, about 0.001 mm. or less in thickness, is demonstrated in adult human temporal bones. The name "barrier membrane of the cochlear aqueduct" is proposed for this structure.

In cases of subarachnoid hemorrhage a pathologic rupture of the barrier membrane caused by a sudden increase of pressure of the spinal fluid seems to precede the hemorrhagic penetration of the cochlea.

Blood may be present in the perilymphatic spaces of the cochlea even if the cochlear aqueduct is completely obliterated by bone.

The fact that blood had been seen at the cochlear entrance of the aqueduct after subarachnoid hemorrhage cannot be accepted as evidence of a physiologic flowing of spinal fluid into the cochlea.

Histologic evidence points to a fluid exchange, if any, that takes place by diffusion through the membranous barrier, rather than to a direct flowing of spinal fluid into the cochlea. Additional experimental studies free of artefacts are necessary to clarify the question of patency of the cochlear aqueduct.

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Case Reports

FOREIGN BODY OF THE MIDDLE EAR

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FOREIGN BODY of the external auditory canal is not uncommon, and an occasional patient is encountered who has succeeded in getting a foreign body into the middle ear by this route. In all cases the foreign body of the middle ear has arrived there by way of the canal except for a few instances of knife wounds and bullets. The foreign bodies found include beads, stones, insects, pencil erasers, various vegetables, buttons, a ball of cotton and bits of metal from welding.[1] The literature on puncture wounds of the mouth and throat is sparse.[2] Innumerable objects have caused injuries of this type, but the injuries usually heal quickly and without complication. A search of the literature, however, has not revealed any instance of a foreign body of the middle ear that had arrived there by way of the mouth.

REPORT OF A CASE

This remarkable feat was accomplished when the patient fell down the front steps of his house onto the sidewalk, on his face. He had been smoking a pipe. When the patient recovered from his initial shock, he found that there was considerable pain in the right side of the face; he was deaf in the right ear; the right side of his face was paralyzed, and several of his back teeth had been knocked out by the injury. He also noticed that his pipe was broken in half and that the stem was missing. After several hours, because of the persistence and severity of the pain, he went to a nearby hospital.

There he received symptomatic medication, and penicillin. A lower molar tooth which remained but which had been loosened by the injury was removed on the ward by a member of the house staff. A biopsy was made of buccal mucosa from the wound of entrance and was reported as showing acute and chronic inflammation. A roentgenogram of the skull was reported as revealing fracture of

1. Faulder, T. J.: Foreign Body in Mastoid Antrum, Proc. Roy. Soc. Med. **14**:26, 1920. Longer, E. P.: Two Interesting Foreign Body Cases of the Mastoid and Nose, Ann. Otol., Rhin. & Laryng. **38**:1158, 1929. Mosher, W. F.: Foreign Bodies of External Canal, Middle Ear and Mastoid and Their Complications, Arch. Otolaryng. **36**:679 (Nov.) 1942.

2. Klimpfel, W.: Puncture Wounds of Mouth and Throat, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:328, 1940. Amano, K. W.: Pipe Stem Impacted in the Tonsil for Fifteen Months, Arch. Otolaryng. **23**:429 (April) 1936.

the right zygoma with undisplaced fragments. Two examinations of the ear were made, and the only finding was that the lumen of the external auditory canal was occluded by clotted blood. The patient had a temperature of 100 F. during the first three days of hospitalization and then remained afebrile for the next two weeks. He was discharged improved, with a diagnosis of (1) fracture of the zygoma, (2) bleeding into the canal of the facial nerve, causing facial paralysis, and (3) cerebral concussion. Two days after his discharge, he succeeded in picking the greater portion of the pipestem out of the muscles of his cheek. Despite this, he continued to have pain, malaise, asymmetry of the face and deafness, so that he applied to, and was admitted to, the Veterans Hospital at Fort Howard, Md.

At the time of his admission he had a low grade fever, a moderate amount of redness and swelling over the right mastoid process and a purulent discharge of the right ear. Penicillin therapy was begun by the officer of the day in the belief that this was acute mastoiditis.

The foreign bodies removed from the patient's ear.

An otolaryngologic examination was made the morning after admission. He had considerable residual inflammation over the mastoid bone. The external auditory canal was markedly narrowed by an inflammatory reaction. The lumen of the canal was filled with nonodorous pus, and when this was removed, an irregular, hard, black foreign body could be seen to be impacted in the swollen canal, partially covered with buds of granulation tissue.

Roentgenograms of the mastoid processes showed them to be large and cellular, with no exudate and no alteration of the normal pattern. A roentgenogram did show some roughening of the superior surface of the condyloid process of the right mandible. The foreign body was radiolucent and could not be demonstrated by the usual routine roentgen view or by one taken with the patient in the Stenver position. No evidence of fracture of the zygoma was seen in these films.

Audiometric examination revealed complete nerve deafness, and the results of tuning fork tests were confirmatory. He had complete paralysis of the affected side of the face. Neurologic examination gave otherwise negative results. In the buccal mucosa just behind the molar teeth could be seen the healing wound of entrance of the foreign body.

After repeated unsuccessful attempts had been made to remove the foreign body through the canal, a radical mastoidectomy was performed by a postaural approach. When the mastoid antrum was opened, the malleus and the incus were found free in the cavity. When the middle ear was exposed, eleven small fragments of the bit of the pipestem were disclosed. The lining of the middle ear was overgrown by a mossy covering of granulation tissue, so that identification or evaluation of the essential pathologic changes was difficult. A Panse flap plastic operation was done. The cavity of the operative wound was packed with gauze soaked in tyrothricin solution, and the skin was closed with interrupted silk Stewart stitches.

The postoperative course was uneventful, and except for a small amount of serosanguinous drainage during the first few weeks, the ear remained dry. The patient has been observed on occasions for fourteen months. During this interval, he has experienced a moderate return of function of his facial muscles, so that his face is symmetric and he is able to close his eyes. His loss of hearing has remained complete. It is interesting that he retained some labyrinthine function. This was discovered accidentally when he experienced vertigo and nystagmus while the ear was being irrigated with penicillin solution that had recently been removed from the refrigerator.

The probable path of the foreign body was through the buccal mucosa, between the medial surface of the ramus of the mandible and the lateral pterygoid plate of the sphenoid bone, grazing the superior surface of the condyloid process, through the anterior wall of the mandibular fossa into the middle ear. The facial nerve was probably injured by the foreign body scraping over its superficial and relatively unprotected course on the floor of the middle ear.

MULTIPLE OSTEOMA OF THE MASTOID CAVITY

WILLIAM A. MARROCCO, M.D.
PATERSON, N. J.

OSTEOMA of the mastoid process is infrequent;[1] multiple osteoma occurring within the mastoid cavity is rare.[2]

REPORT OF A CASE

H. B. a white man aged 39, was admitted to the hospital because of acute nasopharyngitis. On routine examination, a swelling was noticed behind the right ear in the mastoid area (fig. 1). It was not painful or tender to touch; in fact, the patient was not aware of its presence. The auricle was pushed slightly forward. Both external auditory canals were dry and clean. Both drums were intact and had a normal appearance. Hearing was not affected in either ear. There was no history of any infection of the ear or of "ear trouble."

Roentgenograms showed a well developed normal pneumatic mastoid bone on the left side. On the right, there was a large "expanding lesion," the size of the entire mastoid process, containing six separate small osteomas (figs. 2 and 3).

A diagnosis of osteomas of the mastoid process was made, and an operation was performed.

The usual elliptic incision of the skin was made, which revealed an elevation of the cortex of the mastoid bone. Chiseling through the outer table, which was thin and easily removed, one discovered an already existing large cavity in the mastoid process, which gave the impression that a complete simple mastoidectomy had already been performed.

The inner table was pearly white and smooth except for the six small bony tumors, each about the size of a pea and firmly attached by a broad base, arranged in a circle (fig. 4). The bony tumors were removed with little difficulty and no bleeding, leaving a concave base pedicle. There was no pus or fluid in the mastoid cavity. The soft tissue and the skin were sutured tightly, and healing occurred uneventfully.

The pathologic report was as follows: "The gross specimen consists of six masses of bony tissue; the largest measures 1.5 by 1 by 0.5 cm. and the smallest 1 by 0.5 by 0.5 cm. They are grayish white and are markedly irregular in shape.

"Microscopic examination reveals completely necrotic islands of what appears to be ghostlike osteoid tissue.

"Diagnosis: Necrotic osteoid tissue."

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1. Coates, G. M.: Osteoma Growing from the Mastoid Cortex, Arch. Otolaryng. **28**:27-28 (July) 1938.

2. Friedberg, S.: Osteoma of the Mastoid Process, Arch. Otolaryng. **28**:20-26 (July) 1938.

ABSTRACT OF THE AVAILABLE LITERATURE[3]

Osteoid osteoma is a benign neoplasm, which represents a displacement of the normal bone tissue by osteoid tissue and calcified vascular

Fig. 1.—Lateral and posterior views of a patient with multiple osteoma of the right mastoid process.

Fig. 2.—Roentgenographic views of the osteomas occupying the right mastoid cavity.

connective tissue surrounded by a variable zone of sclerosis with or without periosteal new bone formation.

3. Hempstead, B. E.: Osteomas of the Paranasal Sinuses and Mastoid Process, J.A.M.A. **111**:1273 (Oct. 1) 1938. Jaffe, H. L., and Lichtenstein, L.: Osteoid-Osteoma, J. Bone & Joint Surg. **22**:645 (July) 1940. Kleinberg, S.: Osteoid-Osteoma, New York State J. Med. **43**:332 (Feb. 15) 1943.

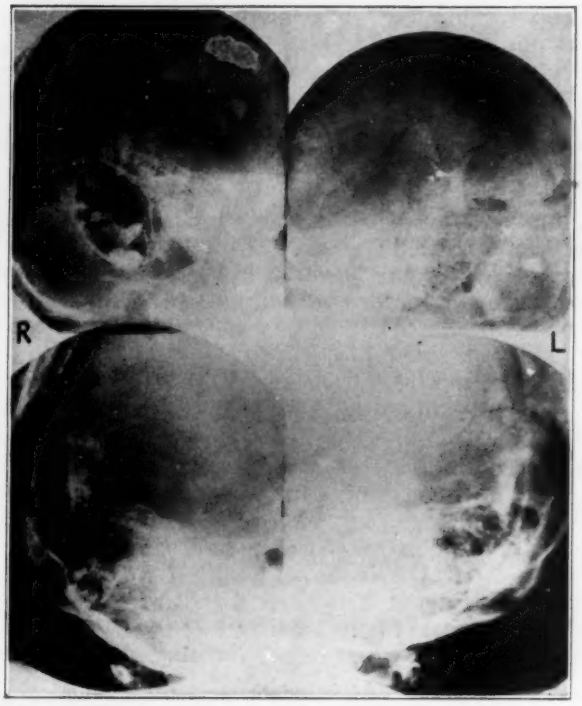

Fig. 3.—Roentgenographic appearance of the involved right mastoid cavity compared with that of the normal left.

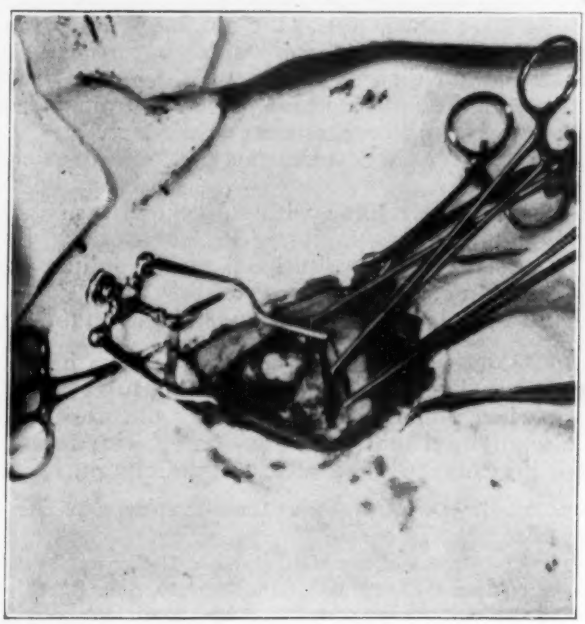

Fig. 4.—This photograph, taken at operation, shows a small osteoma (white object) within the mastoid cavity.

Osteoma of the mastoid should not be confused with exostosis of the external auditory canal.[4] The latter condition invariably arises from the tympanic portion of the temporal bone, and its anatomic situation alone distinguishes it from osteoma of the mastoid process.[5]

Classification.—Gross specimens of osteoma have been classified as:

1. Nonmovable type, with a broad base, firmly attached. This type constitutes the largest percentage.
2. Movable type, sometimes having a pedicle or a pseudarthrosis.
3. Movable, unattached type.

Microscopically, osteoma has been found to be of four types:

1. Osteoma compactum or eburneum.
2. Osteoma cancellare.
3. Osteoma cartilagineum.
4. Osteoma mistum.

Osteoma compactum or eburneum is more frequent than the other types. It is hard, ivory-like, spherical and attached to the cortex of the mastoid process. It may even penetrate into the cells of the mastoid process. Histologically, it shows dense, lamellated bony tissue traversed by a few vessels.

Osteoma cancellare is exceedingly rare. It consists of cancellous bone plus fibrous cellular tissue.

Osteoma cartilagineum is not common and has been reported by many writers. It consists of bone cartilage.

Osteoma mistum is a mixture of the types of bone found in osteoma eburneum and osteoma cancellare.

Etiology of Osteoma.—Tillmanns and Dobbeau stated that these tumors arise from embryonal tissue.

Campbell stated that it is probable that these tumors arise from preosseous connective tissue, just as the normal bones of the head and face form in the embryo without interposition of cartilage.

Simpson stated that there is considerable evidence that the origin of the usual osteoma of the mastoid process is from preosseous connective tissue rather than from remnants of cartilage.

Stuart suggested that the tumor may be due to some pituitary influence.

Friedberg considered trauma with subsequent ossifying periostitis and chronic inflammation as predisposing factors.

Preysse stated that the constitutional and glandular condition of the patient is the cause.

Trauma and infections may have some bearing on the condition.

Diagnostic Observations.—This condition occurs in young adults. The tumor is usually slow growing and produces an external deformity. Pain when present is localized over the site involved. The condition has not been and is not associated with fever. Roentgenographically it presents itself as a relatively radiolucent or rarefied area

4. Simpson, W. L.: Osteoma of the Mastoid, Arch. Otolaryng. **32**:642-661 (Oct.) 1940.

5. Stuart, E. A.: Osteoma of the Mastoid, Arch. Otolaryng. **31**:838-854 (May) 1940. Cinelli, A.: Osteoma Eburneum of the Mastoid, ibid. **33**:421-424 (March) 1941.

of bone, either round or oval, surrounded by what appears as a more or less opaque or dense zone.

Treatment.—Surgical removal is the method of choice and gives prompt clinical cure without recurrence. Complications practically never result from its presence or from its operative removal.

Résumé of Cases Reported in the Literature.—Ten patients had suppurative otitis media in the affected ear; 17 had no suppuration. The macroscopic and the microscopic picture were practically the same in both types, and the final results were also the same in both types of cases.

Probably no connection exists between suppuration of the mastoid process as an etiologic factor and the formation of osteoma.

In 5 of the aforementioned cases there was obstruction of the meatus acusticus externus due to the nearness of the tumor to this canal. The condition was relieved by removal of the growth.

In almost all cases reported the osteoma was single and was found growing external to the cortex.

The sizes varied from that of a small hazelnut to that of a small chicken egg.

COMMENT

This case is reported because there were no cases recorded in the literature available to me in which the osteoma was multiple and located within the mastoid cavity, arising from the inner table of the mastoid process, and not on the external cortex.

445 Park Avenue.

SUBLINGUAL RHABDOMYOMA

T. E. BEYER, M.D.
AND
J. R. BLAIR, M.D.
DENVER

RHABDOMYOMAS are of special interest to the otolaryngologist because of their comparative rarity and because the majority occur in or about the oral cavity.

Parreira and Nunes de Alameida[1] found only 2 cases in a histologic study of 4,720 specimens at the Portuguese Institute of Oncology. Klemperer[2] in a comprehensive review of the literature up to 1933 found 37 cases, in 22 of which the tumor occurred in the tongue. To these 37 cases he added 6 of his own, in 1 of which the tumor occurred in the tongue.

The predilection of rhabdomyoma for the tongue and the oral cavity suggests that trauma may be an etiologic factor. Added weight is given to this view by 4 cases in which the epithelium overlying the tumor was found to be cancerous.[3]

The histology of rhabdomyoma has been exhaustively studied by Abrikossoff,[3a] Dewey,[4] Thoma[5] and others[6] and will therefore not be repeated here. The consensus is that they are usually benign and that metastases are rare.

The diagnosis of rhabdomyoma is usually established only after removal of the tumor, as distinctive clinical and roentgenologic features suggestive of the true nature of the lesion are generally lacking.

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1. Parreira, H., and Nunes de Almeida, J.: Rhabdomyoma of the Tongue, Arq. de pat. **6**:582, 1934; Rhabdomyoma of the Tongue: Two Cases with Review of Cases Previously Reported, Rev. port. de estomatol. **5**:69-85, 1939.

2. Klemperer, P.: Myoblastoma of Striated Muscle, Am. J. Cancer **20**:324, 1934.

3. (*a*) Abrikossoff, J. A.: Weitere Untersuchungen über Myoblastenmyome, Virchows Arch. f. path. Anat. **280**:723, 1931. (*b*) Schirmer, R.: Ueber ein Myoblastom zusammen mit Cancroid der Zunge, Beitr. z. path. Anat. u. z. allg. Path. **89**:613, 1932. (*c*) Derman, G. L., and Golbert, Z. W.: Ueber Unreife aus der quergestreiften Muskulatur hervorgehende Myome, Virchows Arch. f. path. Anat. **282**:172, 1931. (*d*) Dawydow, I.: Zur Frage der unausgeriften Rhabdomyome des Kehlkopfs, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:221, 1931.

4. Dewey, K. L.: Rhabdomyoma of the Tongue, Arch. Path. **3**:645 (April) 1927.

5. Thoma, K. H.: Rhabdomyoma of the Tongue, Am. J. Orthodontics (Oral Surg. Sect.) **27**:235, 1941.

6. Boyd, W.: Surgical Pathology, Philadelphia, W. B. Saunders Company, 1942, p. 733. Ewing, J.: Neoplastic Diseases, ibid., 1940, pp. 240-248.

In the case under discussion a growing tumor under the tongue produced difficulty in mastication and swallowing, which necessitated its removal.

REPORT OF A CASE

A 52 year old railroad conductor first noted a painless swelling under the symphysis of the mandible in the winter of 1944. For the next year this swelling was intermittent and seemingly often associated with exposure. In February 1946 the patient began to experience difficulty in keeping his lower denture in place, and the dislocation of the tongue made mastication, swallowing and even talking increasingly difficult.

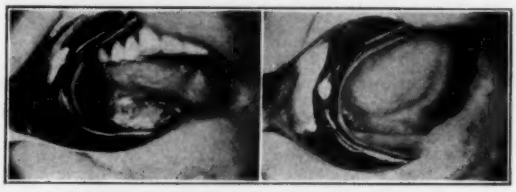

Fig. 1.—Left: Photograph showing the tongue displaced upward and to the right. Right: Postoperative photograph showing the tongue returned to its normal position.

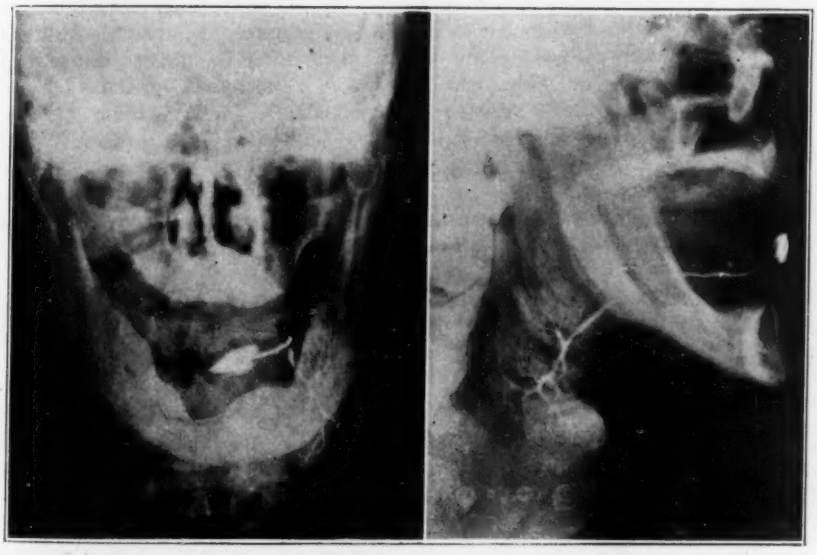

Fig. 2.—Left: Anterior-posterior sialogram showing Wharton's duct displaced laterally by a tumor mass. Right: Lateral sialogram showing an enlarged normal submaxillary gland.

The patient had worn upper and lower dentures for the past twelve years without discomfort.

Except for a train injury to his pelvis in 1919, he had had no previous injuries or serious illnesses.

Physical examination revealed a firm, ovoid, dark red tumor about the size of a large walnut in the floor of the mouth on the left side. The mucosa covering

this mass appeared normal. Wharton's duct was normal in size and caliber. The tongue was displaced upward, backward and to the right (fig. 1, left). The hypopharynx and larynx, owing to the backward dislocation of the tongue, could not be visualized. The left submaxillary gland was palpably enlarged.

A sialogram showed Wharton's duct to be of normal size and caliber and free of foreign bodies, but pushed up and backward (fig. 2). Except for being enlarged the submaxillary gland appeared to be normal.

Repeated incisions of the tumor resulted in brisk bleeding but had no permanent effect on its size or consistency. A course of roentgen irradiation was without appreciable benefit. Accordingly, on June 11, 1946, with the patient under "pentothal sodium" anesthesia, the tumor was removed by blunt dissection. The ends of Wharton's duct which had been torn were approximated, and the wound was closed without drainage. Recovery was uneventful.

The pathologic diagnosis was sublingual rhabdomyoma.

Several weeks after the operation, routine examination disclosed a tumor about 3.5 cm. in diameter in the left hypopharynx which had escaped previous notice.

This tumor was attached in the glossopharyngeal angle and rested on the epiglottis.

On September 4 this was removed by dissection and snare and the left tonsil was removed.

The gross specimen consisted of a tannish brown mass of tissue removed from the region of the tonsil. The mass measured 3 cm. in its greatest dimensions. The sectioned surface was homogeneously tannish brown. Microscopic examination of this nodule revealed it to consist of cells of a large type with a distinct cell wall and a peripherally placed nucleus and in some instances showed distinct cross striations. Among the cells were many thin-walled blood vessels. Fat stains failed to reveal free fat within the cells. Microscopic examination of tissue from the tonsil showed partial to complete fibrous tissue replacement of lymphoid tissue.

The diagnosis was: rhabdomyoma of the hypopharynx, histologically benign, and fibrosis of the tonsil.

Because of persistent enlargement, the left submaxillary gland was removed on September 9. This showed chronic inflammation with fibrosis and atrophy of acini.

When the patient was last examined, Jan. 22, 1947, there was no evidence of recurrence of the tumors (fig. 1, right). He was wearing his dentures comfortably and had no complaints.

SUMMARY AND CONCLUSIONS

Rhabdomyoma in or about the tongue may cause obstructive symptoms requiring its surgical removal.

Roentgen therapy was without curative value in this case.

Sialography proved to be a valuable adjunct in the differential diagnosis.

Fibrosis of the submaxillary gland resulted from prolonged compression of Wharton's duct.

227 Sixteenth Street.

Abstracts From Current Literature

Ear

THE PROBLEMS OF TINNITUS IN THE PRACTICE OF OTOLARYNGOLOGY. ALBERT P. SELTZER, Laryngoscope **57**:623 (Sept.) 1947.

Seltzer discusses some of the theories of the production of tinnitus aurium. The most usual causes are listed. There are many means of treatment, which are discussed. The results are anything but startling, and some modes of treatment are of no value whatsoever. Each case must be studied individually. More emphasis should be placed on treating the person as a whole than on treatment of the head noise itself.

HITSCHLER, Philadelphia.

SOME OBSERVATIONS ON TUMOR OF THE ACOUSTIC NERVE. BERNARD C. MEYER, New York State J. Med. **47**:2436 (Nov. 15) 1947.

Meyer reports on "40 odd cases" from the neurologic services of Mount Sinai Hospital, New York. He refers to the profundity of Cushing's observations, published some nineteen years ago, and adds "some features not mentioned in Cushing's book." These are: increase of spinal fluid protein; hypertension in 12 of 37 (blood pressure above 149 systolic and 90 diastolic); electroencephalographic findings which ranged from normal to a point which suggested focal lesions. On the whole, the last was considered to be "nonspecific." Auditory symptoms preceded all others and were of the classic order, with tinnitus, diminished hearing, vertigo and cerebellar symptoms. Motor twitchings were occasionally confused with jacksonian epilepsy, and were seen in face and eyelid on the affected side. The ages of the patients ranged from 17 to 65. The youngest patient reported by Cushing was 21. In a few patients a loss of the corneal reflex was the only sign of involvement of the trigeminal nerve. Usually there was no facial asymmetry, although the facial nerve was found at autopsy to be compressed or prolongated.

Surgically, the question arises as to intracapsular or extracapsular removal. There are advocates of both methods. Cushing preferred the conservative intracapsular procedure, although he seems to have thought recurrence more likely. The Mount Sinai surgeons found mortality lower and recurrences fewer after intracapsular removal. When total extirpation was done, facial paralysis always followed. Cushing felt that the psychic depression of the patient with facial paralysis was sometimes worse than a recurrence which required a secondary operation.

VOORHEES, New York

ROLE OF BONE CONDUCTION IN THE EFFECTIVENESS OF HEARING AIDS. J. L. VECKMANS, Acta oto-rhino-laryng. belg. **1**:321, 1947.

In cases of transmission deafness the hearing aids tend to amplify too much the low-pitched sounds, with resulting distortion of speech and undue exaggeration of the background noises. The author thinks that attention of the lower tones

and more marked amplification of high tones give better intelligibility; he disagrees with those who hold the opinion that one should realize a selective amplification (i.e., greater amplification of tones definitely lost and less of those better conserved).

HERSON, Chicago.

Pharynx

VITAMIN K AND LATE TONSILLAR HEMORRHAGE. SAMUEL L. FOX AND G. BROOKS WEST JR., Laryngoscope **57**:564 (Aug.) 1947.

Fox and West studied the controversial subject of post-tonsillectomy hemorrhage in relation to the action of vitamin K and that of salicylates, especially a chewing gum containing acetylsalicylic acid.

It is believed that salicylates produce hypoprothrombinemia and that simultaneous administration of vitamin K will prevent that. However, the hypoprothrombinemia produced by even large doses of salicylates was not of a degree sufficient to cause bleeding. The use of the chewing gum did cause increased postoperative bleeding. Vitamin K did not prevent this.

The conclusion was that the bleeding present in the cases in which the patient used Aspergum was caused by some local rather than systemic action.

HITSCHLER, Philadelphia.

POLIOMYELITIS AND TONSILECTOMY. CLAUDE D. WINBORN AND JOHN R. STANSBURY, Laryngoscope **57**:575 (Aug.) 1947.

Additional evidence is submitted concerning the relation of tonsillectomy and poliomyelitis.

The authors conclude that no more of those who had undergone the operation contracted poliomyelitis but that, of those contracting the disease, a greater proportion suffered with the bulbar type. They noted that poliomyelitis occurred in a much smaller proportion of tonsillectomized persons in a nonepidemic period than in an epidemic period.

HITSCHLER, Philadelphia.

PLASTIC REPAIR OF THE PHARYNGOSTOMY. R. J. L. ROBIN, Rev. de laryng. 277 (June) 1947.

The author reviews different technics applied to the pharyngostomy consecutive to Portmann's laryngectomy in three stages. The latter procedure, although requiring protracted hospital care, appears to make possible surgical cure in extensive forms of extralaryngeal cancer and reduces markedly the postoperative bronchopulmonary complications.

The secondary plastic closure of the pharnygostomy is made on the average one to three months after laryngectomy. Depending on the primary incision of the skin, the pharyngostomy is either vertical (i.e., fusiform or oval shaped) or triangular. The fusiform stoma is closed by means of a simple sliding flap from the immediately adjoining skin. The oval and the triangular stoma may be high or low; if high, it is closed by means of the rotating indian flap cut horizontally from the submental region and brought down vertically over the stoma; if low, it is closed by means of a double sliding triangular flap after Imre.

In cases of extensive openings, comprising the whole height of the neck, one uses a combination of the aforementioned procedures—i.e., the indian flap for the upper part of the stoma the first month, and Imre's flap the second month.

In cases in which there is marked scar formation around the stoma one uses a delayed pedicle flap taken from the suprascapular or the pectoral region.

HERSON, Chicago.

Larynx

NEW CONCEPTS OF THE ETIOLOGY AND TREATMENT OF DIVERTICULA OF THE ESOPHAGUS. B. T. KING, Surg., Gynec. & Obst. **85**:93 (July) 1947.

King does not accept the standard explanation that congenital weakness of the pharyngoesophageal wall predisposes to the development of esophageal diverticula. He directs attention to the small ridge at the site of all intervertebral joints and states that the lower border of the cricoid cartilage frequently rests against the ridge of the joint of the fifth cervical vertebra. This ridge continuously pressing against the lower border of the cricoid cartilage causes thinning of the inferior constrictor muscle and the posterior esophageal wall at the point of contact. The upper border of the posterior surface of the cricoid cartilage does not rest against the spine, as does the lower portion, and it is in the latter region that the herniation occurs. The greater muscular development in males, with the consequence that the larynx impinges more firmly on the spine, is said by the author to account for the threefold greater frequency of diverticula among men.

The other concept proposed concerns the closure following amputation of the sac. A narrow blade forceps is placed across the neck of the sac at the point of junction so as not to interfere with or include any of the circumferential wall of the esophagus. Another forceps is placed parallel to the first, and the sac is amputated between the two. Closure is begun with the first forceps, in situ, and a running suture is employed. The forceps is removed and the suture continued to the other side of the amputated sac. The suture must not be pulled to the extent of causing any puckering of the closure. A second similar row then enfolds the first. The resulting closure is preferable to the transfixion and ligation suture, which may cause narrowing or a partial stricture. A one stage operation is advocated.

FRIEDBERG, Chicago.

CARCINOMA OF THE LARYNX: METHODS AND RESULTS OF TREATMENT. G. B. NEW, F. A. FIGI, F. L. HAVENS AND J. B. ERICH, Surg., Gynec. & Obst. **85**:623 (Nov.) 1947.

This article is based on a study of 568 patients treated for malignant laryngeal neoplasms at the Mayo Clinic during a ten year period, 1934 to 1943, inclusive. The difficulty of distinguishing malignant tumors of the larynx from certain benign growths and from tuberculoma or syphiloma on visual inspection alone is mentioned, and biopsy of every laryngeal tumor is held essential. For this procedure, suspension laryngoscopy is used routinely, and frozen sections are obtained. Should the specimen prove unsatisfactory, another piece of tissue can be removed or treated immediately. The grading of malignancy according to Broder's classification is a most important factor in the selection of treatment in each case of laryngeal carcinoma.

Four methods of treatment are employed: (1) thyrotomy (laryngofissure) and surgical removal of the growth, (2) laryngectomy, (3) removal or treatment of the local lesion under suspension laryngoscopy and (4) external irradiation. In deciding which is the most suitable method of therapy in the individual case one considers size, the location and the extent of the laryngeal growth, the grade of malignancy, the pressure or the absence of extralaryngeal extensions or of metastases involving cervical nodes, and the age and the general physical condition of the patient.

Complete removal of the neoplasm is the prime consideration in any of the operative procedures, but unnecessary sacrifice of the voice should be avoided; a radical operation should not be employed when a conservative one will remove the neoplasm equally well and will leave the patient with a good functional voice; nor should a conservative operation be used when a more radical procedure is indicated. The authors consider that it is unjustifiable to try a conservative form of treatment on the assumption that, if the results are not good, something else can be attempted later.

The type of tumor which is ideal for thyrotomy (laryngofissure) is of low grade malignancy and is confined to the anterior two thirds of a vocal cord which is freely movable. The fact that a growth extends across the anterior commissure does not preclude the possibility that it may be extirpated by thyrotomy.

All patients who have intrinsic laryngeal carcinoma with fixation of one or both cords but no evidence that the lesion has metastasized to the cervical nodes are subjected to laryngectomy, provided their general condition will permit. However, unless extremely small, a high grade carcinoma involving a vocal cord should be treated by laryngectomy, even though there is no cord fixation. This statement is based on the fact that active epithelioma may often extend far beyond the apparent limits of the growth.

A small low grade epithelioma of vocal cords which are not fixed can be removed by surgical diathermy under suspension laryngoscopy, with good results. This statement applies to a small group of selected cases. If the neoplasm cannot be brought under direct view in its entirety or if there is any question as to the extent of the growth, thyrotomy should be performed. A well circumscribed and low grade malignant tumor of the epiglottis, the aryepiglottic folds or the base of the tongue can be treated by electrocoagulation under suspension laryngoscopy. Often after removal of a malignant tumor the base of the lesion is treated by insertion of radon seeds. Preliminary tracheotomy is carried out in all these cases.

Irradiation is reserved for high grade, radiosensitive lesions that are too extensive to warrant their being treated by surgical measures. It is employed infrequently by the authors. Preliminary trachectomy is performed whenever intensive roentgen therapy is to be used.

Of the 568 patients in the series, 78.5 per cent were treated by surgical methods and 21 per cent by irradiation. Of the patients who underwent surgical treatment and could be traced, 73.5 per cent survived five years or more without recurrence. In the laryngectomized group 60.2 per cent had a five year cure; 83.6 per cent of the traced patients who underwent thyrotomy lived five or more years; in the group treated by surgical diathermy or insertion of radon seeds, under suspension laryngoscomy, 91.7 per cent lived five or more years. In the group treated by irradiation, only 6.9 per cent showed a five year survival. The authors admit that the low survival rate of patients treated by irradiation is to be expected, since practically all the patients had inoperable lesions which were considered hopeless when first examined.

FRIEDBERG, Chicago.

ERRORS AND DIFFICULTIES IN EXTRACTING FOREIGN BODIES FROM THE BRONCHUS AND THE ESOPHAGUS. C. FERNANDEZ F., Rev. de otorrinolaring. **7**:22 (March) 1947.

The author presents a group of 5 patients who had a foreign body either in the trachea or in the esophagus The purpose of the presentation was to illustrate some of the difficulties and complications that arose in the treatment of these patients. These difficulties were occaisoned by a number of factors: 1. Removal of the foreign body was attempted by an untrained person. 2. In several cases faulty and improper instruments were employed. 3. Extreme carelessness was exhibited in the failure to localize the foreign body. In several of the cases there was lack of a preoperative roentgen examination. 4. Negligence arose in the postoperative follow-up studies. In one of his patients a large retropharyngeal abscess developed twelve days after a bronchoscopic examination. 5. There was failure to employ the proper anesthetic in a number of the cases reported.

The presentation is worth while, since it is a frank indictment of members of the medical profession, for there are times when physicians become hasty, overzealous and overconfident in meeting these acute critical problems. This is an excellent warning that each case must be carefully studied, evaluated and followed to complete termination with the greatest amount of care.

PERSKY, Philadelphia.

Nose

NASAL HEMORRHAGE. H. E. HARRIS, Eye, Ear, Nose & Throat Monthly **26**:81 (Feb.) 1947.

A method is presented for the control of the severe type of epistaxis, which usually originates in the posterior half of the nose and usually requires packing of the anterior and posterior parts of the nose.

A small curved metal suction tube is covered with a tight-fitting rubber catheter, with the metal tip protruding slightly at the distal end. A short wave electrosurgical unit is then used, and, with one electrode attached to the patient's skin, the circuit is completed by touching the other electrode on the exposed metal at the proximal end of the suction tube. In this manner blood is aspirated rapidly from the bleeding area, which can then be located and the bleeding vessel electrocoagulated. It may be necessary to slow the bleeding temporarily by inserting a pack saturated with a 10 per cent solution of cocaine hydrochloride containing epinephrine hydrochloride.

By this method, nasal hemorrhage of the massive severe type has been controlled in 43 cases during the past seven years. Ligation of the internal maxillary or the external carotid artery has not been found necessary. A short discussion of the blood supply of the nose is included.

JENNES, Waterburg, Conn.

USE OF NEW AGENTS IN THE TREATMENT OF EPISTAXIS. B. A. COPE and M. M. HIPSKIND, Eye, Ear, Nose & Throat Monthly **26**:417 (Aug.) 1947.

The gelatin sponge and oxidized cellulose are utilized in the treatment of epistaxis to avoid causing discomfort to the patient by the use of bulky packing. Since these agents do not have to be removed, the possibility of fresh nasal hemorrhage on removal of the packing is obviated.

The bleeding point is visualized with the help of gentle suction with a capillary tip. A local anesthetic agent may be used. A small gelatin sponge is cut to

a size which will allow it to cover the site of the bleeder and at least 3 to 5 mm. extra surface around the site of bleeding. This pack is then introduced over the bleeding area and is held in place for a few moments while the edge of the sponge is tamped down to the mucous membrane. The pack usually disappears in about four to five days, either by being absorbed or by being picked or blown out by the patient. It is sometimes difficult in the Kesselback-Little area to get the sponge to stick, but in these cases of nonadherence it can usually be found that part of the sponge is on the cutaneous margin of the columella.

Where pressure is also needed, as in some cases of bleeding from the posterior portions of the nose, oxidized cellulose is used. In the most common sites of epistaxis, the gelatin sponge is preferable, both from ease of application and rapidity of action in controlling the bleeding.

CHANGING CONCEPTS OF SINUSITIS. F. JOHNSON PUTNEY, Laryngoscope **57**:664 (Oct.) 1947.

Putney reviews the present concepts of sinusitis. He classifies cases of sinusitis as allergic, mechanical and bacterial, and discusses each class.

NEW ACQUISITION OF MEDICINE IN THE PROCESS OF RHINOSINUSITIS. ALEJO P. BELOU, Rev. argent. de oto rino-laring. **15**:338 (Sept.-Oct.) 1946.

The author presents a detailed description of the problems encountered in the treatment of rhinosinusitis. He groups these under (1) aeration and ventilation of the nasal cavities; (2) destruction or inhibition of the germs; (3) reaction of the body, and (4) protection of the useful mucous membrane and elimination of the degenerated mucosa.

He describes the pathologic process of the mucous membrane of the nose and accessory sinuses. He discusses the various medicaments commonly employed in shrinking the edematous mucous membrane to afford proper aeration and drainage. Difficulties arising in a speedy cure are often due to many anomalies of the structure of the various sinuses and of the nose.

In dealing with the inhibition and destruction of the various organisms, the author enters into considerable detail in regard to the various sulfonamide compounds, stressing their evolution from the earliest products to the present accepted preparations that are more commonly employed. He describes the various methods of treatment, including both the parenteral and the local application. Locally he commonly uses the displacement method of Proetz after the preliminary decongestion of the mucous membrane. Instilling of solutions of sulfonamide drugs into the larger sinuses, such as the frontal, sphenoid and maxillary sinuses, has definitely shortened the course of infection and lessened the dangers of complications. However, emphasis is placed (1) on danger of the clinical symptoms and (2) on the danger that some germs may become sulfonamide resistant and may continue to produce symptoms of a low grade nature which can flare up on the slightest provocation. The contraindications to the use of sulfonamide drugs are hepatic infections and severe blood dyscrasias. The role of penicillin and its effectiveness are discussed. This antibiotic is administered both parenterally and locally. The author recommends that 10,000 to 20,000 Oxford units be given by intramuscular injection every three hours in cases of acute disease. More recently the action of penicillin has been prolonged through the use of a preparation of the substance in oil. Locally the penicillin can be instilled into either the maxillary or the frontal sinuses in doses of 3,000 to 5,000 units. This instillation may be repeated every three hours, and when this procedure is used, the author

employs an indwelling catheter or cannula. In the treatment of the acute or the subacute infections of the ethmoid sinus he employs the Proetz technic. Some of the untoward effects of penicillin are dermatitis, allergic reaction, fever and chills, various generalized pains, including headaches, muscular pains and colicky pains, and, finally, transitory azotemia.

The author also recommends that tyrothricin be used locally in the sinuses—an antibiotic which has given splendid results, particularly in cases of osteomyelitis.

To increase the general resistance of the patient, a diet is stressed, since in sinus infections, particularly during the acute stage, there is an increase in the basal metabolism, and hence the caloric intake should be increased. There are often a nitrogen disturbance, a water imbalance and a decrease in the normal functioning of the digestive tract. The author recommends a diet high in calories and an increase of the vitamin intake, stressing particularly ascorbic acid, and an increase of the mineral intake, but advises that the diet be of a bland nature, so that the digestive tract may function freely. The author always recommends the use of autogenous vaccines, particularly in the cases of chronic infection. Elimination of any allergic factor is extremely important.

Surgical treatment is indicated purely to preserve a normal mucous membrane and to remove any part of the mucous membrane that has undergone permanent pathologic changes. In conclusion the author makes the following observations: 1. Chemotherapy and antibiotic medication are efficacious factors for the control of the sinus infections; 2. Failure to obtain good results is due principally to incomplete study of the patient and indiscriminate use of medication. 3. Careful bacteriologic study is important, so that the proper medication can be employed for the destruction of the organisms. 4. At present the most satisfactory treatment includes the use of penicillin. 5. It is necessary to increase the natural defenses of the body by a proper dietary regimen and vitamin therapy. 6. It is necessary to establish a normal state of the mucosa and proper drainage of the cavities. 7. Antibiotic medication is the best means of curing the sinuses, but surgical intervention may still be necessary to clear up many irreversible pathologic conditions in the sinuses.

PENICILLIN THERAPY IN SINUSITIS. SANTIAGO A. ARANZ AND ALFREDO R. CORDERO, Rev. argent. de oto-rino-laring. **16**:1 (Jan.-Feb.) 1947.

The authors outline their method of administering penicillin in the treatment of acutely infected sinuses. They administer penicillin both locally and parenterally. Locally they instil penicillin into the maxillary sinuses, the frontal sinuses or the sphenoid sinuses as indicated by the pathologic conditions Their method is to insert into the sinus either a cannula or an antrum puncture needle and permit it to remain in situ during the entire period of treatment. At regular intervals the sinus is irrigated through this cannula or needle with about 100 cc. of water containing 2,000 units of penicillin. The treatment continues as long as the suppuration persists. The solution is retained in the sinus cavity for at least one-half hour. Treatment is repeated every three or four hours except for the fact that with the earlier treatments the authors used from 10 to 20 cc. of solution. The only difficulties encountered with this method of treatment are as follows: Occasionally, when the walls of the sinuses are extremely thick, it may be difficult to introduce the needle or the cannula; a mild ulceration may occur on the septum near the tip of the nose or on the inferior border of the nostril; dermatitis may be caused by the adhesive tape which holds the cannula in place; occasionally there may be obstruction of the needle or the cannula.

Penicillin is also administered parenterally in adequate doses; i.e., about 5,000 units per cubic centimeter of solvent is injected intramuscularly at the usual three hour intervals.

In a series of 82 cases, maxillary sinusitis was treated in 67, bilateral in 13, frontal sinusitis in 8 and pansinusitis in 2. The total quantity of penicillin usually amounted to 500,000 units per patient. The average length of treatment was five days. Within the first twenty-four hours there was an appreciable decrease or complete disappearance of pain, which was due to the better drainage and aeration of the sinuses. There was also a marked improvement in the general condition of the patient, including a definite fall in temperature. In conclusion the authors state in regard to the acute infectious process that complete cure was affected in 100 per cent of the cases. When there was an acute exacerbation of a chronic infection, the acute state was immediately cleared up but the chronic lesion was unaffected. In chronic infections of the sinuses the treatment did not produce any favorable results.

PERSKY, Philadelphia.

OSTEOMYELITIS OF THE FRONTAL BONE. ATILIO VIALE DEL CARRIL, Rev. argent. de oto-rino-laring. **16**:17 (Jan.-Feb.) 1947.

The author describes the mechanism of infection and osteomyelitis of the frontal bone and some of the problems associated with both the pathologic conditions, the complications and the treatment. Under treatment he stresses the importance of early extensive surgical exenteration not only of all the visible pathologic tissue but also of apparently normal bone beyond it. This treatment should be supplemented with the administration of both sulfonamide drugs and penicillin. The latter can be used locally; a solution containing 1,000 units of penicillin per cubic centimeter can be instilled through a drainage tube into the operative area. Parenterally penicillin is given in doses of 20,000 to 30,000 units every three hours. Since these newer drugs have appeared, the prognosis of ostomyelitis has improved immeasurably, and the therapeutic problem depends on two factors: (1) curative (surgical treatment associated with both parenteral and local administration of penicillin and sulfonamide drugs, and local application of tyrothricin) ; (2) prophylactic (prolonged desensitization with autogenous vaccines).

PERSKY, Philadelphia.

Miscellaneous

PLASTIC SURGERY IN INDUSTRY. C. L. STRAITH AND M. A. PILING, AM. J. SURG. **74**: 328 (Sept.) 1947.

The authors' method of treating a crushing injury of the nose is to elevate the depressed bones with a flat instrument, such as a chisel or a periosteal elevator, then to support them by packing the nasal cavity with petrolatum gauze. A special apparatus is described and illustrated, for maintaining fixation until the fragments are firmly united. For correction of a saddle nose, preserved homologous rib cartilage is used, together with methods of rhinoplasty not notably different from those in general use.

PLASTIC OPERATION FOR HARELIP PROFILE. A. RITHI, J. Internat. Coll. Surgeons **10**:381 (July-Aug.) 1947.

When harelip is associated with a curved nose, as it often is, the author corrects the nose first and then shortens and thickens the upper lip. The technic is carried out with the patient under local anesthesia. Seven drawings and photographs serve to demonstrate the excellence of his operative results.

ALBERT P. SELTZER, Philadelphia.

SPIROMETRIC EVALUATION OF BETA-DIMETHYLAMINOETHYL BENZHYDRYL ETHER HYDROCHLORIDE (BENADRYL) IN ASTHMA. L. LEVY II and J. H. SEABURY, J. Allergy **18**:244 (July) 1947.

Spirometric methods afford a simple and reliable means of judging the quantitative effects of various drugs used in the treatment of asthma. Subjective evaluation of any drug given to the asthmatic patient is of little scientific significance. Spirometric studies were performed on 16 patients thirty minutes after oral administration of 100 mg. of diphenhydramine hydrochloride ("benadryl hydrochloride"). When compared with the initial spirometric tracings, those obtained after treatment showed no consistent changes in vital capacity, tidal air, minute ventilation, expiratory differential, respiratory rate or degree of emphysema.

After epinephrine and aminophylline had been administered to 5 of these 16 patients, there was a uniform increase in vital capacity, tidal air, minute ventilation and expiratory differential and no increase in the respiratory rate.

Six of the patients derived subjective benefit, with decrease in dyspnea, from the use of "benadryl." The spirometric data for 3 of these were directly opposed to the subjective report. In 1 patient the response obtained was the same as that observed after the administration of epinephrine and aminophylline.

Since epinephrine and aminophylline afford both subjective relief and objective spirometric evidence of improvement in most asthmatic patients, the authors feel that these drugs should be used as a base line by which newly introduced drugs for which bronchodilator effects are claimed may be quantitatively evaluated.

FRIEDBERG, Chicago.

"PYRIBENZAMINE" VERSUS SPECIFIC HYPOSENSITIZATION IN THE TREATMENT OF POLLINOSIS: A COMPARATIVE STUDY. C. E. ARBESMAN, V. L. COHEN and H. OSGOOD, J. Allergy **18**:311 (Sept.) 1947.

The treatment of ragweed hay fever with tripelennamine hydrochloride ("pyribenzamine hydrochloride"; N′ pyridyl=N′ benzyl = N = dimethylethylene=diamine hydrochloride) alone was, in the authors' experience, as effective as the usual injection treatment for the relief of symptoms in clinic patients. Injection therapy plus the use of the usual symptomatic drugs, such as iodides, ephedrine and aminophylline, proved superior to the administration of tripelennamine hydrochloride alone in the alleviation of bronchial symptoms. There is no question regarding the value of antihistaminic drugs as used in the treatment of ragweed or hay fever. Tripelennamine hydrochloride alone, in sufficient dosage, may control the symptoms of seasonal allergic rhinitis to a great degree, but it cannot give as adequate relief as does proper hyposensitization plus administration of tripelennamine hydrochloride. The incidence of side effects of this drug is low when the drug is given with specific hyposensitization, because smaller doses are required to control the symptoms.

FRIEDBERG, Chicago.

THE INTELLIGIBILITY OF CONNECTED DISCOURSE AS A TEST FOR THE "THRESHOLD FOR SPEECH." G. A. FALCONER and H. DAVIS, Laryngoscope **57**:581 (Sept.) 1947.

An interesting test is described for measuring loss of hearing. It consists in measuring the threshold of intelligibility of connected discoure (TICD). The patient listens to monitored connected discourse. He himself adjusts the loudness to the point at which he is just able to understand everything said. The setting is recorded.

The test is quick (two minutes) and reliable (it was compared with the standard test for threshold of speech hearing, in which monosyllabic words are used); it is interesting and not fatiguing to the patient. Its chief disadvantage is the subjective nature of the determination of the end point. Few, however, recorded erroneous thresholds.

This test should prove to be of great clinical value.

HITSCHLER, Philadelphia.

THE USE OF ANTIBIOTICS IN THE TREATMENT OF BACTERIAL INFECTIONS. W. BARRY WOOD JR., Laryngoscope **57**:657 (Oct.) 1947.

Penicillin is effective against gram-positive micro-organisms (particularly the gram-positive cocci). It is effective in the treatment of syphilis and in that of certain highly fatal diseases, such as pneumococcic meningitis, empyema, subacute bacterial endocarditis, acute osteomyelitis and thrombosis of the cavernous sinus.

Streptomycin is effective against gram-negative bacilli, such as the colon bacillus, "Friedländer's bacillus and Hemophilus influenzae, and in tularemia. Streptomycin produces more toxicity than penicillin. Vertigo and deafness have been encountered frequently.

The pharmacologic aspects, the dosage and other principles of antibiotic therapy are discussed.

HITSCHLER, Philadelphia.

REGIONAL SPEECH DIFFERENCES IN VETERANS AND NONVETERANS. BURTON H. BYERS, Quart. J. Speech **33**:312 (Oct.) 1947.

Twenty-three nonveterans, mostly women, and 28 veterans, mostly men, ranging in age from 17 to 35 and in classification from freshman to junior at Henderson State Teachers College, were observed for variations from the general American dialect. The results showed an increasing elimination of the more easily recognizable dialectic differences on the part of the veterans. The author states that further study would probably indicate a correlation between length of service and the modification of speech differences, and between general intelligence and the extent to which regional differences were modified.

PALMER, Wichita, Kan.

A STUDY OF ENCEPHALITIS. MILDRED GOTTDANK and DORIS TREPEL, Quart. J. Speech **33**:355 (Oct.) 1947.

This article comprises a review of the medical background of encephalitis and a report of a case in which an ambulatory patient with chronic encephalitis was aided in the speech clinic for eighteen months. The symptoms of "a postencephalitic adult" coming to the speech clinic will be a combination of one or more of

the following: (1) tremors; (2) drooling; (3) a masklike expression; (4) bizarre posture; (5) definite symptoms of a disturbance of speech. A patient with chronic encephalitis finds himself burdened with physical, social, vocational and economic problems, to which are added, for the most part, the additional ones of vocal and articulatory difficulties. In the case presented, the patient showed tremor of the left arm, masklike expression, infrequent blinking, stooped posture and uneven gait. The voice was monotonous, lacking variation in pitch and rate, and was almost inaudibile. The rhythm was irregular, and the patient seemed to find difficulty in verbalizing his thoughts. There was apparently poor lip and lingual mobility which made articulation sluggish, although there was good control of lingual and labial muscles. The breathing was somewhat shallow, and the entire speech pattern lacked animation. The patient appeared frightened during interviews, and it was impossible to put him at ease. He stated he had always needed speech training but had never had the courage to go to a clinic for help. The technics used consisted of practice of reading exercises, use of the voice mirror, and development of good phonation. When phonation became habitual, lip and tongue exercises were begun, the patient being encouraged to articulate clearly individual sounds and later words, phrases and sentences. After this, work with pitch, rate and intonation was begun. Question and answer practice was carried out many weeks. Finally the subject was placed before a mirror, and many facial expressions were tried out and used. When he was discharged, emotional and psychologic integration was at least well on its way to successful completion. On discharge he was able to speak of his difficulty with ease and objectivity and was free from destructive self consciousness. He still had a fixed expression to some extent, occasional difficulties of thought and, during fatigue, immobility of the lips. Both clinic and patient were satisfied with the results. It seems reasonable to believe that there are many other ambulatory patients with chronic encephalitis who could benefit by speech reeducation.

PALMER, Wichita, Kan.

CONTRIBUTIONS FROM THE MILITARY PROGRAMS IN VOICE COMMUNICATION. WILLIAM B. MCCORD, Quart. J. Speech **33**:370 (Oct.) 1947.

The material developed from the work done by the Army Air Forces in voice communications and speech intelligibility is useful to the public as well as to the speech clinic for improving speech communications in noise. In general the following factors and suggestions were found to be most effective: 1. Speak loudly, but don't shout. 2. Use a moderate tempo, prolong the sounds, especially in important words or phrases. 3. Group words into short phrases—don't talk word by word. 4. Emphasize in a steady, natural manner but limit the variation of pitch, loudness or tempo. 5. Speak distinctly; work to be understood. 6. Make your speech sound natural and meaningful. 7. Learn to use your particular communication equipment with maximum efficiency. (*a*) Speak directly into the mouth piece, receiver or microphone, with the lips lightly touching it. (*b*) If using ear phones, wear them directly on the ears. (*c*) Turn the volume up as high as possible when the voice signal is weak. B. Practice listening in noise. 9. Develop standardized procedures if possible, using standardized nomenclature and pronunciations.

Many of the teaching programs in the military services were widely acclaimed for their remarkable accomplishments. Much of the rapid learning was due to the unusual motivation based on (1) the urgency of the situation, (2) the controlled, concentrated study, and (3) the clarification of goals.

CANCER AND POLITICS; THE OPERATION ON GROVER CLEVELAND. M. G. SEELIG, Surg. Gynec. & Obst. **85**:373 (Sept.) 1947.

In 1893 one of the most dramatic surgical operations in American history lent color to a period that was packed with sufficient explosive tinder to alarm the best and most patriotic minds of our people. The patient was Grover Cleveland, the President of the United States. The operation was the removal of the left side of the upper jaw, which was the seat of cancer.

Shortly after Cleveland won his second election for the presidency, a number of economic factors were responsible for great unrest in the country. Heavy gold exportations by the United States, the suspension of free coinage of silver by India, the lack of confidence in public credit due to the gold shortage in the treasury, the fear of a silver basis, unwise speculation and general unemployment all made for critical times. A disastrous panic occurred on June 27, 1893. President Cleveland had called a special session of Congress for Aug. 7, 1893, to repeal the Sherman Silver Purchase Act.

On June 18, 1893, Dr. R. M. O'Reilly, who later became Surgeon General, examined the President and discovered an ulcer of the left side of the hard palate. A biopsy was pronounced to be highly suggestive of cancer by the pathologist of the Army Medical Museum (the name of the patient had been kept secret). Dr. Joseph D. Bryant, of New York, was called to see the President and advised an immediate operation. Every circumstance connected with the incident was shrouded from both the members of Congress and the public at large. Dr. W. W. Keen was also called in consultation.

At different times of the day and the night of June 30, 1893, all parties concerned boarded a private yacht lying in New York harbor. The utmost secrecy surrounded the proceedings. On the morning of July 1, with the yacht sailing at half speed up the East River, an operation was performed. Under nitrous oxide anesthesia the two upper left bicuspid teeth were extracted. Ether was then substitued and, with Drs. Keen and Erdmann assisting, Dr. Bryant removed the entire left side of the upper jaw from the first bicuspid to beyond the last molar tooth. The antrum was found to be partly filled by a gelatinous mass. Bleeding was controlled with pressure, hot water and the galvanocautery.

The operation required approximately one hour, and an estimated 6 ounces (150cc.) of blood were lost. The President was out of bed on the second day after the operation. The pathologic diagnosis was sarcoma. On July 17 another operation was performed because of the fear that remnants of the tumor had escaped removal. The defect of the palate was corrected by means of an artifical jaw made of vulcanized rubber.

On August 5, just twenty days after the second operation, the President arrived in Washington to direct the strategy of the repeal of the Sherman Law. Authorities in the field of history are said to concede that, had Cleveland's opponents been aware of his physical condition, the gravity of the national consequences would have been unpredictable.

Grover Cleveland died fifteen years after the removal of his jaw. The cause of death was recorded as "heart failure, complicated with pulmonary thrombosis and oedema." It is known that Mr. Cleveland had suffered from a gastrointestinal disorder and that he died of intestinal obstruction. It has been impossible to determine whether death was due to abdominal cancer. If it was, one might speculate whether the lesion was an independent primary growth or a metastatic lesion. The latter possibility is extremely doubtful.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

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Regular Monthly Meeting, Dec. 1, 1947

Benign Cysts of the Paranasal Sinuses. Presented by DR. HAROLD F. SCHUKNECHT.

Benign cysts of the paranasal sinuses are of two general types, those which take origin from the sinus mucosa and those which invade the maxillary sinuses from the teeth. From the mucosa arise three types: The nonsecreting cysts, the mucous gland (retention) cysts and the mucoceles.

Nonsecreting cysts are found in the maxillary sinuses and occur in about 5 per cent, as shown by routine roentgenograms. On roentgenograms they produce a characteristic dome-shaped shadow, but may also fill the sinus and produce complete homogeneous clouding. The cyst forms as a collection of straw-colored fluid in the loose subepithelial connective tissue of the mucosa. This fluid is usually sterile and coagulates on standing. Small nonsecreting cysts usually produce no symptoms and need not be removed. When the cyst occupies one half or more of the sinus, there may result not only local symptoms but general symptoms of fatigue, irritability and headache. Having no epithelial lining, this cyst may disappear after spontaneous rupture or after puncture and lavage. Or cure may be obtained by an antrum window operation, with part or all of the cyst wall being removed through the window. Ossacionally a radical removal of the contents of the sinus is necessary.

The mucous gland (retention) cysts occur in all the sinuses. They are small, not individually evident on roentgenograms, have an epithelial lining and appear as part of the histopathologic picture in chronic hyperplastic sinus mucosa. Hyperplasia of the epithelium of the duct of the tuboacinose gland is probably an important factor in producing stenosis with resulting retention of secretion and cyst formation. At the time of operation these small cysts are often misinterpreted as being small abscesses.

Mucoceles occur commonly in the frontal and ethmoid sinuses. Obstruction of the nasofrontal duct or of a particular ethmoid ostium probably occurs in all cases of true mucocele. Repeated acute infections, in some instances accompanied by temporary blockage, appear to increase the probability of eventual permanent blockage and mucocele formation. As the products of secretion, inflammation and desquamation accumulate in the cystic space, the latter expands to encroach on the nasal cavity, the orbit and the anterior cranial fossa. The cyst invades the orbit early by pressure erosion of the floor of the sinus, with characteristic proptosis and lateral and downward displacement of the eye. Acute infection of a mucocele (pyocele) may be a serious complication if the cyst has expanded into the anterior cranial fossa. Chronic mild infection of a mucocele may result in chronic hyperplastic osteomyelitis, in which case the cystic space may be almost filled with new bone. An intranasal operation may be adequate; however, usually a radical external frontal-ethmoidal sinusotomy is necessary. An opening into the nose must be created which will remain open.

Odontogenic cysts are of two types: the follicular cyst and the radicular (root) cyst. The follicular cyst arises from the enamel organ epithelium of the dental follicle. When one or more teeth are present, it is commonly termed a dentigerous cyst. The cyst will not contain a tooth if it develops from the enamel epithelium before this has become sufficiently differentiated to form enamel. The lining epithelium is stratified squamous in type and closely resembles oral epithelium. As the cyst expands, it may invade and fill the maxillary sinus. If a tooth is present in its wall, it may be displaced into the sinus. The radicular cyst develops from the apical or root granuloma. Epithelial cell rests of the periodontal membrane may be included in the root granuloma and by inflammatory stimulation may completely line the central cystic space as it forms. Radical removal of the sinus contents is necessary for both types of odontogenic cysts, for which the Caldwell-Luc operation is generally best suited.

DISCUSSION

DR. WALTER THEOBALD: I wonder if many of these cysts do not get well with simple lavage. I have found that after irrigation and finding a cyst present, with return of straw-colored fluid, on later irrigation the cyst wall itself may wash out the opening or into the middle meatus so that it can be removed. I think, furthermore, that the window operation does suffice in the majority of cases for removal of these cysts.

DR. SAMUEL SALINGER: What is the significance of the simple inclusion cyst so far as the patient is concerned? It has been my experience that in most cases it causes little trouble and is often discovered accidentally on routine roentgen examination. Frequently it disappears sponstaneously and occasionally is punctured during irrigation of the antrum. In my opinion it is not significant unless it attains sufficient size to block the ostium. The case of the mucocele is entirely different; this should be removed. The question as to what procedure is best is a matter for another discussion.

DR. JOHN R. LINDSAY: These nonsecreting cysts have interested me for some time, probably because I have encountered a few which were not so benign as had been thought. Cultures of the straw-colored fluid were usually sterile. My associates and I made a chemical study of these fluids a few years ago; the fluids have a high protein content, corresponding to exudates rather than transudates. When they are small, perhaps less than half filling the sinus, they are probably innocent. Yet I have had the experience of puncturing one cyst with a lumbar puncture needle and withdrawing about 10 cc. of fluid, and the next day finding the patient in bed with acute arthritis. I have had other experiences in which simply letting the fluid out relieved acute pain in the region of a joint. In other instances there was no evidence that the cyst was producing symptoms. Some will disappear after irrigation. They do not have an epithelial lining and may collapse and disappear. Others recur and require an antrum window operation to permit removal; still others require radical antrotomy for adequate removal. I have observed cases in which a window operation had to be followed by radical exenteration of the mucosa of the sinus to prevent recurrences and relieve the accompanying distress. When there is evidence of much hyperplastic change of the mucosa, in addition to the cyst, it is better to do the radical operation than to risk having to do a second operative procedure.

DR. MAURICE F. SNITMAN: Does Dr. Schuknecht include cystic adenoma among the benign cysts of the upper jaw or the maxillary sinus?

DR. SHERMAN SHAPIRO: Do these benign cysts ever undergo cancerous degeneration?

DR. HAROLD F. SCHUKNECHT: Dermoids frequently involve the nasal structures, but rarely the paranasal sinuses proper. I think the only type that becomes cancerous is the follicular cyst. Kahn, in 1933, reported several cases in which the follicular cyst apparently developed into adamantinoma.

Fundamental Changes of Concepts in Regard to Chronic Suppurative Sinusitis. Presented by DR. HENRY L. WILLIAMS, Rochester, Minn.

In the last twenty-five years more fundamental changes have taken place in the organized body of concepts that form the philosophy of chronic suppurative disease of the paranasal sinuses than in any other division of otorhinology. These conceptual changes fall into two groups. The first group includes those concerning the clinical pattern of chronic suppurative disease of the paranasal sinuses. Formerly, chronic suppurative sinusitis was thought to be characterized by severe pain in the head or headache, and long papers were devoted to the description of the headache pathognomonic of suppuration of certain particular sinuses; it was considered to be due to pressure of retained secretion on the wall of the sinus.

As a result of investigative work by Thomas Lewis, Kellgren, H. G. Wolff and his co-workers, and Behnke, it is now generally recognized that, as a rule, chronic suppurative sinusitis is a painless disease. Exceptions that "prove the rule" do occur, and it is important to recognize them clinically.

When chronic suppurative sinusitis co-exists with clinical evidence of chronic vasomotor rhinitis, however, the condition is quite different. This combination of disorders I term "chronic hyperlastic sinusitis." The clinical pattern is characterized by prominence of the symptom of headache. Because patients who have allergic involvement of the paranasal sinuses without symptoms and signs of suppuration are seen in whom the symptom of headache is a prominent clinical feature, and because pain in the head (or headache) is an unusual clinical feature in patients with uncomplicated chronic sinusitis, it seems reasonable to conclude that headache in a patient with chronic hyperplastic sinusitis results from the allergy rather than from the suppuration. By clinical examination it can then be ascertained that the headache does not arise from the sinuses but usually from other pain-sensitive structures about the head, as a rule the cranial and cervical musculature and the extracranial blood vessels. It would appear, then, that to secure clinical relief equal attention must be given to both the allergic and the suppurative factors.

The so-called routine allergic management of hyperplastic sinusitis has proved disappointing. Positive results of skin tests rarely give specific information, generally indicating only that the patient under consideration is pathergic; attempts at obtaining relief by specific desensitization usually fail. In chronic hyperplastic sinusitis, diagnosis by means of elimination diets also has proved ineffective. The author believes these failures to result from the fact that in this syndrome what is presented generally is not an antigen-antibody type of reaction but, rather, so-called physical allergy, for which the term "pathergy" has been suggested by Urbach. This disturbance appears to be failure of the general adaptation reaction described by Selye, the normal physiologic process proceeding no further than the stage of "alarm." Because of cellular injury, histamine is released in the alarm reaction, producing a clinical picture indistinguishable from that of "allergy."

It is imperative that the allergic treatment of a patient with such sinusitis be carried out by, or in close association with, the rhinologist. So-called nonspecific allergic treatment is usually more successful in relieving the allergic symptoms than so-called specific methods. Among nonspecific methods are included subcutaneous injection of foreign proteins, such as typhoid bacilli and milk, and the

use of house dust. The low dosage method of Hansel is favored. Symptomatic relief has been obtained for a number of patients by the use of capillary vasodilators, such as monoethanoleamine nicotinate ("nicamin"), sodium nicotinate, nicotinic acid, histamine, neostigmine, magnesium sulfate and the like. "Nicamin" is preferred because of ease of administration.

Certain rhinologists maintain that there is an allergic factor present in nearly all cases of chronic suppurative sinusitis. At the Mayo Clinic, microscopic examination of sections of mucosal lining and other tissue removed at operation in cases in which chronic suppurative sinusitis had been diagnosed almost invariably revealed an inflammatory picture, thus confirming the so-called allergic picture. The role of the eosinophil in allergy is unknown, and it is questionable if its presence in examined tissue is a sufficient basis for an unqualified diagnosis of allergy.

Formerly, it was felt that free and adequate drainage of the rigid-walled cavities was the prime consideration, and to this end the functioning structures of the nasal chambers, the mucosa and the turbinal tissues were freely sacrificed. Thirty years ago Lillie was one of the first to protest against this indiscriminate surgical practice. Under his guidance, physiologic investigations were undertaken by Hansel, Stark, Heetderks, Hilding and others. Proetz, Van Alyea and others have added greatly to the knowledge of nasal function and its importance to well-being. It is now generally accepted that while adequate drainage must be secured, even more important is preservation of the functioning nasal tissues. It is now generally recognized that operative procedures should be adequate to achieve drainage and aeration, and at the same time rational enough to preserve the functioning structures of the nose. In the condition defined as chronic hyperplastic sinusitis a judicious combination of surgical and allergic treatment must be used to give symptomatic relief.

Several technical changes in operations on the various sinuses have resulted from this changing philosophy. There is a trend toward use of the nasoantral window in operations on the maxillary sinus; in most instances removing the lining mucosa is contraindicated. The ethmoid sinuses are almost invariably involved along with other sinuses; in simultaneous involvement of the maxillary and the anterior ethmoid sinus, a nasoantral window frequently is sufficient to relieve both disturbances. In suppuration of the frontal and ethmoid sinuses, however, exenteration of the ethmoid sinuses is necessary to afford access to the frontal sinus. Chronic frontoethmoid suppuration, in my opinion, does not lend itself to preservation of the mucosa of the frontonasal duct as proposed by Van Alyea. In most instances reactive osteitis tends to produce progressive constriction of the frontonasal duct, occasionally resulting in formation of a pyocele. As a rule, therefore, intranasal frontoethmoid exenteration is the procedure of choice, though external frontoethmoid exenteration requires less skill and experience and is potentially less dangerous. Another reason in favor of the intranasal procedure is the greatly diminished tendency for scar tissue to close the nasofrontal opening, one of the principal causes of failure in external operations on the frontal sinus. This is due to wide destruction of the frontal process of the nasal bone and removal of the lamina papyracea of the ethmoid bone, as usually practiced in the Lynch technic. The first procedure allows collapse of soft tissue into the approach to the frontonasal opening, while removal of the lamina papyracea favors formation of excessive granulation tissue from the periorbita, with subsequent closure of the frontonasal opening by scar.

External operation on the frontal sinus is justified when the disease has perforated one of the walls of this sinus with formation of an abscess or a fistula.

also when there is evidence of partitioning in the frontal sinus by bony laminas, or in cases in which reactive osteitis has so hardened the bone in the area that dangerous force would be necessary in performance of an intranasal procedure.

Continuance of the external incision around the inner canthus of the eye favors involvement of the venous plexus around the lacrimal sac, the importance of which in promoting spreading osteomyelitis of the frontal bone and osteitis of the facial bones has been pointed out by Lillie. With the use of tantalum foil, shaped to fit the frontonasal opening at the time of operation, I have found that closure of this aperture can be avoided.

DISCUSSION

DR. O. E. VAN ALYEA: The society is indeed fortunate to have Dr. Williams give his ideas about the changing philosophy and conception of sinus disease. I agree heartily with almost everything he has said. All who do much work in the fields of rhinology and laryngology are becoming "allergy minded." As is often the case, however, when a new idea takes hold, there is a rush to put it into practice, and it becomes a fad with many people who overlook the wide range management of the conditions concerned. The reports from dust desensitization have been amazing. Yet are not otolaryngologists somewhat inclined to regard every patient as allergic, overlooking some other fundamental factors in the disease and in the symptom complex?

In surgical procedures on the frontal sinus, Dr. Williams displays a leaning toward intranasal methods when possible. Of course, it has always been consistent to include with procedures on the frontal sinuses exenteration of the anterior ethmoid sinuses. I have never been able to understand why the whole group of ethmoid cells needs to be sacrificed. It is true that certain cells en route to the frontal sinus, frontal cells or ethmoidofrontal cells, near the ostium, are of major importance. In my opinion one should give up the idea of removing bullar cells; they never block the frontal sinus, and one disturbs an area which may be free of infection. However, cells which do block drainage must be removed, by passage of a cannula or otherwise. Those in the lumens of the sinuses may be taken care of through the external frontal approach without disturbing the sinus or the ostium.

During the past few years my associates and I have studied some old sections removed by radical operations in such cases and find, amazingly enough, that the sections obtained in these cases contained a preponderance of eosinophils. The results of surgical intervention were temporary; many of the patients are now wrecks due to chronic nasal disease. Those of them that we still see occasionally in the dispensary are trying to get relief from symptoms that are now worse than they were before resort was made to radical surgery.

Penicillin in my hands has been a failure as administered by the Proetz method and I now reserve it for patients that have serious symptoms, for which it is indicated.

Dr. Williams did not mention the "irreversible" changes referred to constantly in the literature. I have not encountered any. My experience has been similar to his; when the antrum is opened by a window procedure and thickened membrane is found, recovery takes place when drainage is established. Is this an irreversible change? One hears frequently of little abscesses between the sinus and the bone. In our examination of specimens we failed to find any abscesses in extirpated mucosa, nor did we find ulcers and other conditions that have been described.

Another item Dr. Williams failed to mention is the so-called ethmoiditis. We see conditions of multiple polyposis of the middle meatus; yet on examination of specimens from the anatomic laboratory, polyps in the middle meatus are usually seen to arise from the maxillary ostium, the uncinate process and the bulla. I have yet to find one in which polyps could be traced to the ethmoid labyrinth.

DR. SHERMAN SHAPIRO: I should like to ask Dr. Williams if he considers that complete obliteration of the frontal sinus can be achieved by any method short of removal of the entire anterior wall and anterior floor, including the septum. I should also like to ask if there is any postmortem material at the Mayo Foundation or elsewhere showing that obliterated frontal sinuses have remained so.

DR. SAMUEL SALINGER: To the older men in this audience this subject is of extreme interest because they have seen a remarkable evolution in the general concept of sinus disease; the younger men know only half the story. The other half is buried in textbooks, voluminous literature and the memory of "has-beens." I well recall when discussions were confined solely to surgical procedures, with argument as to which was best. The volume of surgical treatment of the sinus was tremendous. One member of this society reported that he had performed intranasal frontal sinusotomy with the rasp three hundred times. My own predecessor, Dr. Otto Freer, was strong for a motor-driven burr with special curets of his own. The Halle operation was widely employed, often with disastrous results. Then came the external frontoethmoid exenteration, which was extensively employed and of which one hears little of late!

The trend to conservatism has been definite and unmistakable. Year by year in my own practice I do fewer and fewer operations, and so-called radical sinusotomy is becoming almost as rare as the simple mastoidectomy. Increasing knowledge of the physiology of the nose and of the constitutional factors giving rise to nasal symptoms have explained many of the bad results which formerly followed operation.

I agree with Dr. Williams that allergy studies have often proved disappointing except in certain types of cases. We have found the results more significant in children and adolescents than in adults, probably owing to nutritional and endocrine factors.

There is one item I should like to stress: The time to begin treatment of chronic sinusitis is when the acute attack subsides. Otolaryngologists are prone to dismiss patients too soon. It is important at this time to check the sinuses thoroughly, ascertain their functional capacity and examine the patient for factors which might predispose him to future attacks with increasing damage to the lining mucosa.

I should like Dr. Williams, if he will, to state more explicitly just what he considers the proper indications for intranasal antrostomy.

DR. MAURICE F. SNITMAN: In a study of more than 200 specimens of nasal mucosa removed during radical sinusotomy my associates and I found typical chronic inflammation present in only 2 instances. In most cases the microscopic picture was that of edema associated with varying degrees of eosinophilia.

DR. G. H. MUNDT: It seems to me there are some indications for entering the maxillary sinus through the canine fossa. It is a simple procedure and one gets a much better view than by other means. There is little reaction, general or local, and one can make a satisfactory opening into the inferior meatus without destruction of the inferior turbinate. I have long since discontinued trying to remove all mucosa from the maxillary sinus.

I should like to ask Dr. Williams to speak on antihistamine drugs.

DR. ROBERT H. FRASER: There is much sinus disease in Michigan, and people have to have something done. I have been greatly helped by Dr. Williams' delineation of principles and by this rich presentation. All are groping toward better understanding. Selye and Williams have pursued the defects of adaptation as existent in both old and young.

The surgical procedure which my associates and I follow consists of certain steps in the course of progressive screening processes. In regard to the maxillary sinus the number of smears showing eosinophils is revealing; next comes roentgen examination and, if needed, diagnostic irrigation. The more severe suppurations, even some of dental origin, seem, in my hands, to yield to irrigations in which 2 cc. of a saline solution of penicillin (10,000 units per cubic centimeter) is used, given ordinarily at six day intervals until the mucosa is normal. The empyema may be at an end, and in time its concomitant derangements, with the result that allergy succeeds further. At this point iodized oil reveals such pedunculations (irreversible) as sway the selection of needed surgical intervention from an antral window to a canine fossa approach, or even to the Caldwell-Luc operation. I believe the necessity of surgical intervention must be faced, although the number of patients operated on in the twenties has been screened to the point of decimation—screened surgery becoming conservative surgery.

With Dr. Williams one must respect the problems of those cases of chronic frontal sinusitis which necessitate external operation with, perhaps, employment of the Riedl collapse type of obliteration. I should like to recall the presentation of Dr. W. Likely Simpson, of the University of Tennessee College of Medicine, given before the Academy in October 1943. He has had a large and remarkably successful series through employment of the Lynch approach, which his presentation showed to lead to prompt and permanent healing, including obliteration of the cavity on the blood clot principle. I can vouch for the preoperative data and for the satisfactory healing that occurred in a considerable number of his cases. Credited as integral to the success are a number of Simpson requirements. The elimination of mucoperiosteal remnants in the course of both the frontal sinusotomy and the complete exenteration of the adjacent ethmoid cells is precisely executed in steps which one might call "minute and meticulous." The operator works much under mirror check and through mirror vision within the sinus to achieve finished surfaces. The newer detail includes a motor-driven burr for irregularities. The nasofrontal duct is entirely removed. The middle turbinate is left intact. Little packing is used and that only in the nasal fossa for twenty-four to forty-eight hours, after which no treatment is carried out except slight cleansing of the lumen of the nose. The frontal cavity is not disturbed after operation. With all the floor of the frontal sinus removed and an exenterated ethmoid labyrinth, the inferior aspect of the operative field is disregarded, with neither expansion nor constriction of whatever outlet is then present, granulations not being feared. The sinus area is expected not to be reentered at any time. Penicillin, since it has become available, is administered for seven or eight days, furthering the blood clot method, with successful organization of the clot, in order to obtain obliteration of the cavity. I know of a case of an anteroposterior depth of 18 mm. which, by this procedure, was managed without incident. I am sure Dr. Simpson has made valuable contributions which are capable of formulation and practical application.

DR. HENRY L. WILLIAMS: I do not know that a diagnosis of allergy is justifiable in every instance in which a large number of eosinophils are found in the mucosa on histologic examination. The role of the eosinophil in allergy is not known. Code and others have suggested that the eosinophil is the principal source of histamine, but I do not believe the finding of many eosinophils in the

nasal mucosa on microscopic examination unqualifiedly indicates that the patient has an allergic disease; in an occasional case there could be reasonable doubt. The histologic or the cytologic diagnosis of allergy cannot always be substantiated by a trial of routine allergic methods of treatment, or of antihistamine drugs, or of other nonspecific antiallergic treatment. It seems to me, therefore, that the mere presence of eosinophils in nasal smears or in histologic sections is insufficient evidence on which to base a diagnosis of allergy.

The same statement can be made about polypoid formation. Polyposis results when fluids escape from the blood vessels into the pericellular tissues and edema results. Where underlying tissue is deficient in fibrous and elastic elements, the edematous part is not supported, hangs down and forms a polyp; that is not hyperplasia. As Dr. Galloway has said, "in the occasional case when sufficient aeration and drainage are given" evidence of allergy disappears and the appearance of the nasal mucosa returns to normal. If one were an allergist, one might argue that bacterial allergy produced the polyps which disappeared on eradication of the infection. It is difficult to prove that hypothesis one way or the other.

It can be shown that allergic symptoms do not always demonstrate that a child is allergic. Children fed an abnormal diet sometimes have symptoms of vasomotor rhinitis which are not on an allergic basis. Dr. Arild Hansen, of Minneapolis, indicated the role of lack of unsaturated fatty acids in producing this symptom. The rhinitis sometimes clears up on administration of unsaturated fatty acids. If children eat fat meat, they will not have this type of nasal congestion; but if they will not eat it, a teaspoonful of lard mixed with sugar and spice given as candy three times a day will relieve the symptoms and give them their quota of fatty acids. Butter has a low iodine number and cannot be substituted. Corn oil is good. Edema of the nasal mucosa may be associated with a low basal metabolic rate in both adults and children, and oftentimes is the only symptom observed. The use of thyroid relieves the symptoms, which indicates, I think, that the symptoms are not allergic. I am not one of those who insist that the basis of all chronic suppuration in the sinuses is allergy.

In regard to operation on the frontal sinus, Dr. Van Alyea appears to be more conservative than I am when he advocates saving of the cells of the bulla ethmoidalis. I believe the easiest and best pathway to the frontal sinus by the intranasal route is through the bullar cells. I usually remove not only the bullar cells but those of the agger nasi as well, and enlarge the nasofrontal opening laterally, anteriorly and posteriorly. The floor of the frontal sinus should be removed to the septum and laterally as fas as possible without breaking into the orbital tissue. In my experience this usually results in a permanent intranasal opening. My preference for the intranasal operation on the frontal sinus, when feasible, lies not so much in preserving the mucosal lining—that is important—but in preventing the collapse of the soft tissues which occurs when their support is removed as in the external procedure. Dr. Simpson advocates an external approach to the infected frontal sinus with removal of the frontal process of the superior maxillonasal bone and the orbital plate of the ethmoid bone, much as the late Dr. Lynch did. I believe this widespread removal of bone in the entrance to the frontonasal opening is poor technic.

In regard to Dr. Van Alyea's proposal to preserve the frontal duct, I have not seen many cases in which the nasofrontal duct was not involved in disease of the frontal sinus. In many cases I have found constricting reactive osteitis in this region. In many cases pyocele does not seem to be due to infection of previous mucocele but is produced by bony growth constricting the nasofrontal openings. I do not mean to imply that pyocele never is a secondary result of mucocele but

that it is, I believe, in many instances the direct result of infection and the effect of this on the bone.

Dr. Van Alyea did not obtain good results from intranasal instillation of penicillin. I agree if instillation is done by the Proetz method once a day by the doctor. Instillation of 2 cc. of a solution of penicillin into a sinus does not seem to accomplish more than lavage with isotonic solution of sodium chloride. The sinusitis of an occasional patient will appear to be cleared up by lavage with penicillin solution or even with saline solution. The results of instillation of penicillin solution can be improved if the patient applies it himself several times a day by Hymes's modification of the Proetz technic. The poor results from penicillin therapy in my experience were due to the fact that in order to secure therapeutic effect the solution of penicillin had to be maintained in contact with the mucosa for two or three hours. Apparently ciliary activity takes place even in the presence of serious disease, so that a solution introduced but once daily is removed before sufficient time has elapsed to produce a result. If the solution is introduced six or eight times a day by the patient himself, in certain instances a curative effect is produced. I do not say cure of patients is achieved in 100 per cent, but cure in as many as 10 per cent is a relatively good result.

By hyperplastic ethmoiditis I mean nasal disease in which allergy and infection are mingled. While it is true that, as Dr. Van Alyea states, many polyps hanging in the nose do not originate in the ethmoid labyrinth, yet the cells in the ethmoid labyrinth are full of polypoid material. The postoperative application of radium, which tends to produce fibrosis, and therefore increases the supporting structure in the tissue, is beneficial. Radium is not curative but is worth while, as it gives symptomatic relief and does tend to prevent recurrence after removal of polypoid tissue.

Dr. Shapiro asked whether unilateral ablation of the frontal sinus can be achieved. I believe it can be done if care is taken to eradicate the mucosa from, and bevel the edges of, the frontal sinus as originally suggested by Lillie. Proof that the sinus never reforms is difficult to get because patients do not wish to submit to an examination unless symptoms recur. However, I have a patient who has been without symptoms for more than five years after this type of operation. It seems to me that the poor cosmetic result of ablation is not really an important consideration now that cosmetic procedures have improved. Furthermore, if a patient has been operated on several times with recurrence of infection after each procedure, he does not care how he looks if he can get rid of the infection, and for such a patient I think no other type of operation is indicated. After the infection has been absent for six months or a year, suitable reparative plastic work can be done. The coronal incision is valuable in an ablating procedure if the patient has not been operated on previously with resulting unsightly scarring. This incision gives more adequate exposure and allows better beveling of the superior and lateral bony margins of the sinus, so that no dead space remains. Ablating the mucosa of the sinus and then allowing fibrosis to destroy the lumen of the sinus, I believe, cannot be depended on to destroy the lumen. I have known cases in which secondary abscesses occurred in the center of the fibrous tissue, with considerable discomfort and pain. I think there are many failures with this technic.

Dr. Salinger asked about indications for nasal antrostomy. I feel this operation should be carried out when infection in the antrum fails to clear up after one or two lavages, or when a patient after so-called cure with lavage has repeated recurrence of antral disease. If the presence of an infected root of a tooth is ex-

cluded, the patient gets prompt objective and symptomatic relief by nasal antrostomy. It is a satisfactory operation.

Dr. Mundt stated that he secured good results in chronic maxillary sinusitis with the Caldwell-Luc technic without removal of the lining mucosa. I think this is true. The approach I employ is different, but the end result is the same. If one is more skilled in the Caldwell-Luc technic and gets better results with it, I do not see why one should not use it. It is a little more extensive and requires longer hospitalization than some other procedures. Whether an intranasal window or a Caldwell-Luc approach is made, the end result depends on the adequacy of the window left behind.

Antihistaminic drugs merely give symptomatic relief; they are not a cure. As soon as administration is stopped the symptoms return. For that reason they are unsatisfactory as a rule, because allergy is a lifelong affliction. Good initial results are obtained from administration of 50 mg. four times a day of almost any antihistaminic drug. As soon as the edema of the nasal mucosa subsides, the polyps recede, unless secondary fibrosis of the polypoid tissue is present. After the maximal result is obtained, I decrease the dose gradually in an attempt to find the smallest possible dose that will give symptomatic relief. One reason for the failure of allergic therapy is the lack of recognition of physical allergy, for which the precipitating stimuli are changes in temperature, emotional storms, etc. Antihistaminic drugs are helpful temporarily, but a more important use for them is in rapid diagnosis of an allergic or pathergic condition.

Dr. Fraser spoke of the use of iodized poppyseed oil for diagnosis and of penicillin for treatment of chronic sinusitis. At the Mayo Clinic iodized oil was tried by Dr. Pattee for diagnostic purposes in a large group of patients who gave a history suggestive of sinusitis. Roentgenograms were made at intervals. Twenty-four hours after instillation of the oil there was tremendous swelling of the lining membrane of the sinus. Roentgenograms made after instillation of iodized oil did not appear to have much additional diagnostic value as compared with those made by the usual method.

The etiologic problem of the polyp originating in the antrum and coming into the nasal chamber through the natural ostium has not been solved. This type has a tendency to recur unless the mucosa at the site of origin is removed. Such a polyp occurs in a single antrum; the other paranasal sinuses do not show evidence of polyposis. A large choanal polyp protruding into the nasopharynx has been mistaken for nasopharyngeal fibroma. At the Mayo Clinic histologic study of these polyps and their mucosal attachments has not suggested a definite cause for their appearance even though search has been made for a neoplastic tendency in the sinal mucosa.

Book Reviews

Das akustische Trauma. By L. Ruedi and W. Furrer. Price 26 Swiss francs. Basel, Switzerland: S. Karger.

This is a profound and enlightening study of the complex problem of acoustic trauma. The alliance between the otologist and the physicist proves its value throughout the monograph. The student of acoustic trauma obtains valuable information from the physical definitions, the noise spectrums, the oscillograph recordings, the pressure curves and the frequency analysis of explosions.

The biologic response of the ear was studied in clinical observations, animal experiments and histologic examinations of the ear. Two groups of aural traumas are described. The first group is composed of professional deafness, report trauma of small arms and blunt injuries of the head without fracture of the temporal bone. The symptom complexes of this series are similar in spite of the diversity of causes. Typical is the C-5 dip; in early cases it is the only finding, but later it widens to a broad perception type deafness.

In the second group are aural damages created by explosions of grenades, bombs and heavy artillery. Typical of the latter group is the extensive damage done to the ear drum and structures of the middle ear. Some of the middle ear deafness is reversible. The damage of the inner ear is permanent. The C-5 dip is not found, but vestibular dysfunction is frequent. Unusual is the author's observation of rupture of the basilar membrane and hemorrhage involving the perilymphatic spaces without rupture of the ear drum.

The experimental part centers around two otologic problems:

First, what are the pathologic changes in the ear due to explosions and what is the correlation with the function of the ear? Second, what is the origin of the C-5 dip?

Guinea pigs were exposed to acoustic trauma, and the effect on function was determined by the pinna reflex. The histologic alterations, which are clearly portrayed, were correlated with the functional results. Of interest is the fact that Reissner's membrane was seen to be mildly damaged. The authors show a great interest in the nature of the C-5 dip and offer a hydrodynamic hypothesis as an explanation. On the basis of Bekesy's concept of an eddy formed in the inner ear, they assume that the direction of the eddy changes with frequency. The shift from left turn to right turn occurs, in their opinion, at a place which is the representation of C-5. The influence of fatiguing tones was found to be different when the fatiguing tone was above or below 4096. The details of this interesting observation should be seen in the original.

A device protecting against acoustic trauma is described. The protection afforded by the ear defender is demonstrated on quinea pigs and on workers exposed to industrial noises.

A physiologist may criticize the use of the pinna reflex as an indicator of cochlear function. The use of conditioned reflexes would have been a superior, though much more complicated, method. There are 126 pictures, curves, audiograms and photomicrographs which, owing to their clear print and concise legends, contribute greatly to the understanding. This book is an excellent contribution.

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